# THE American Journal OF Gastroenterology

VOL. 28, NO. 1 JULY, 1957

Panel Discussion on Diseases of the Gallbladder and Pancreas

Chylothorax and Chylous Ascites

Management of Gastrointestinal Bleeding

Massive Hemorrhage of the Upper Gastrointestinal Tract

***Twenty-second Annual Convention***
***Boston, Massachusetts***
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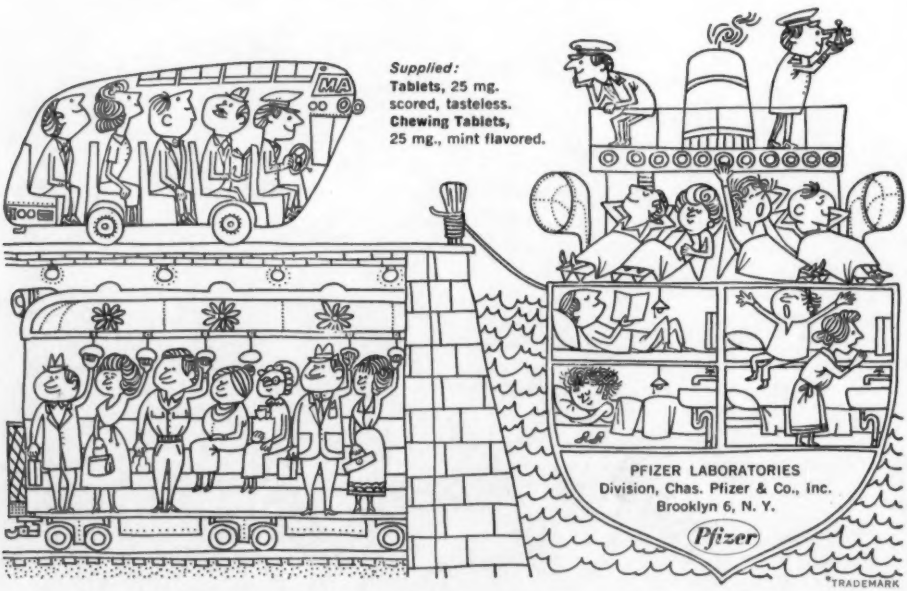

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*The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada*

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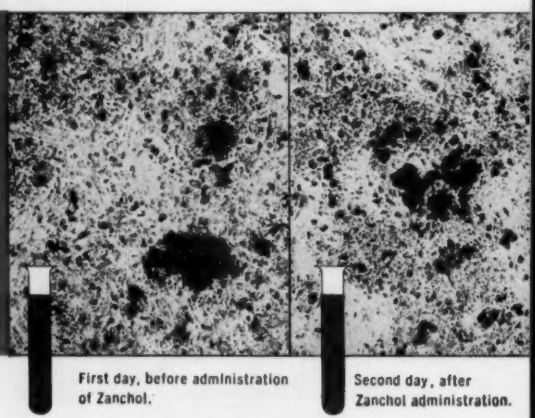

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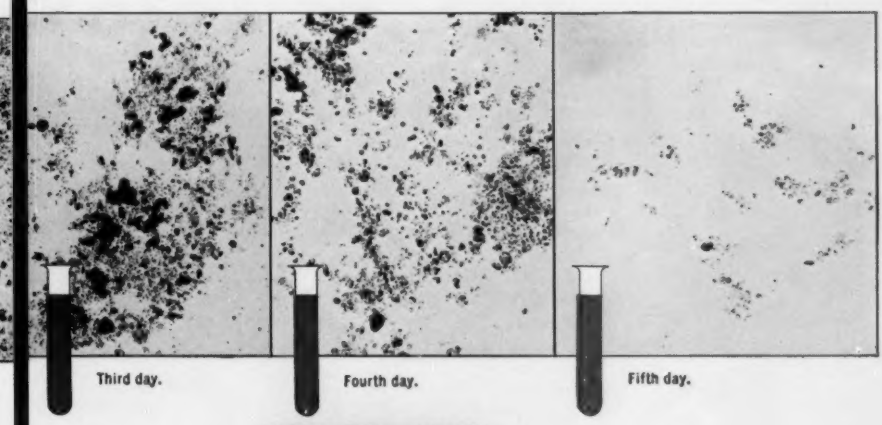


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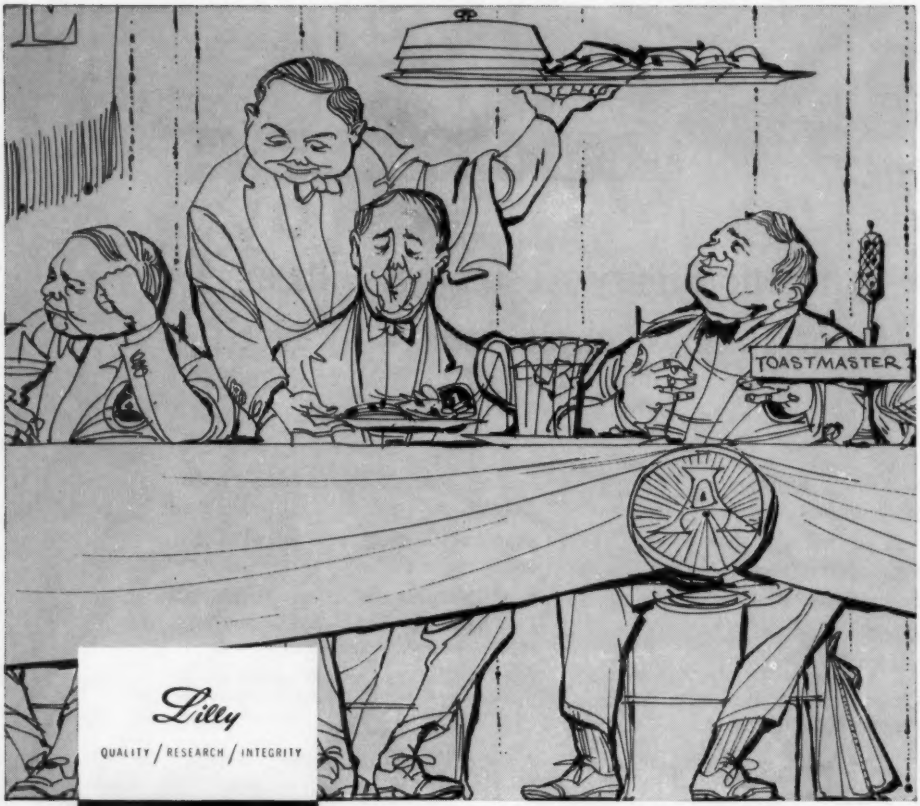


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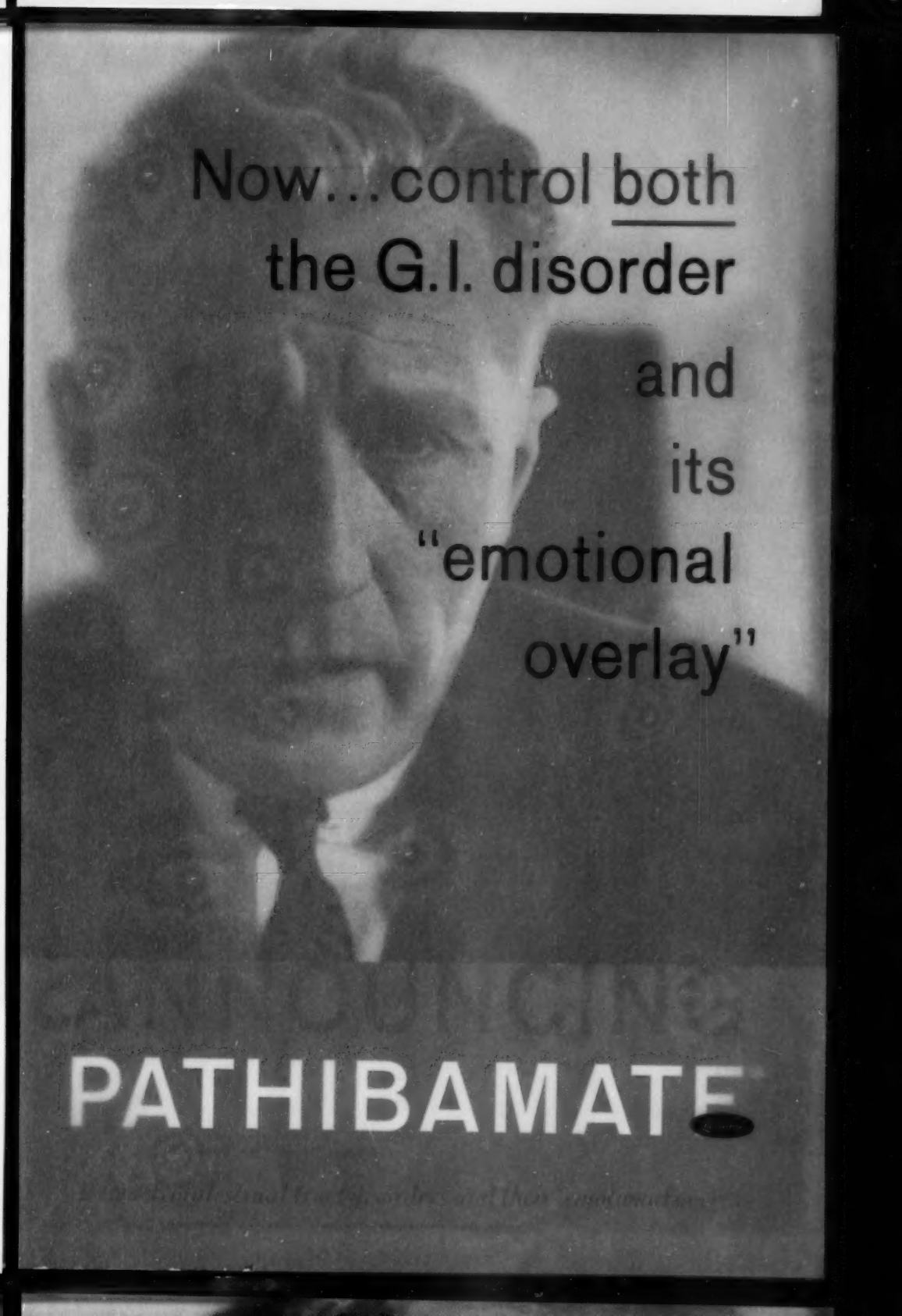
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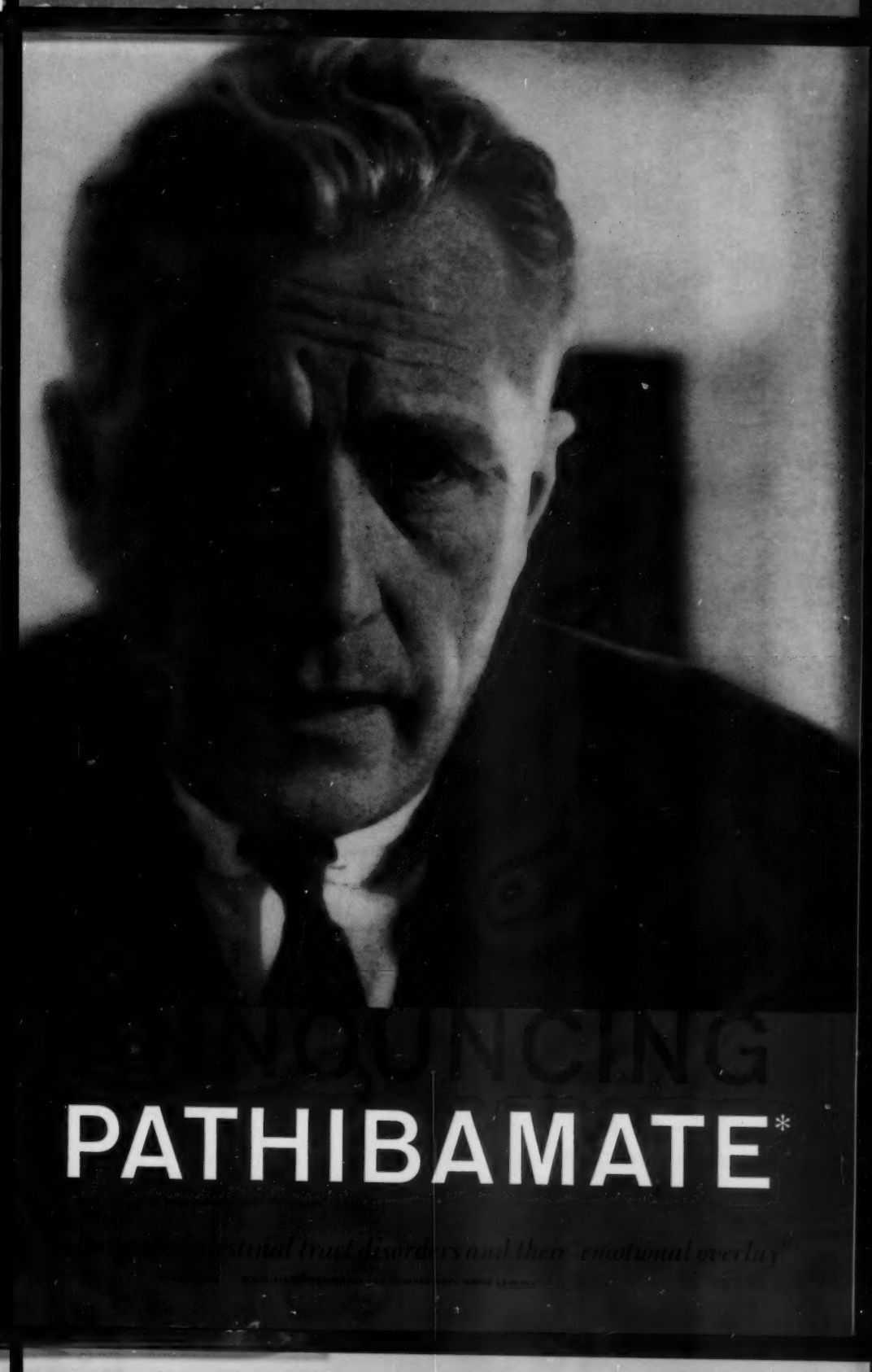
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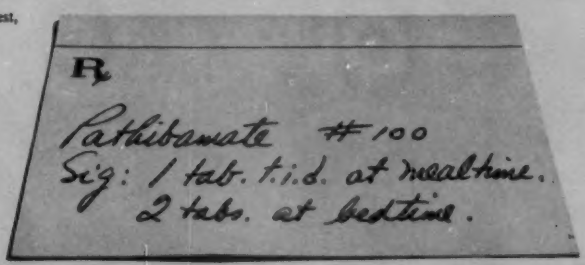


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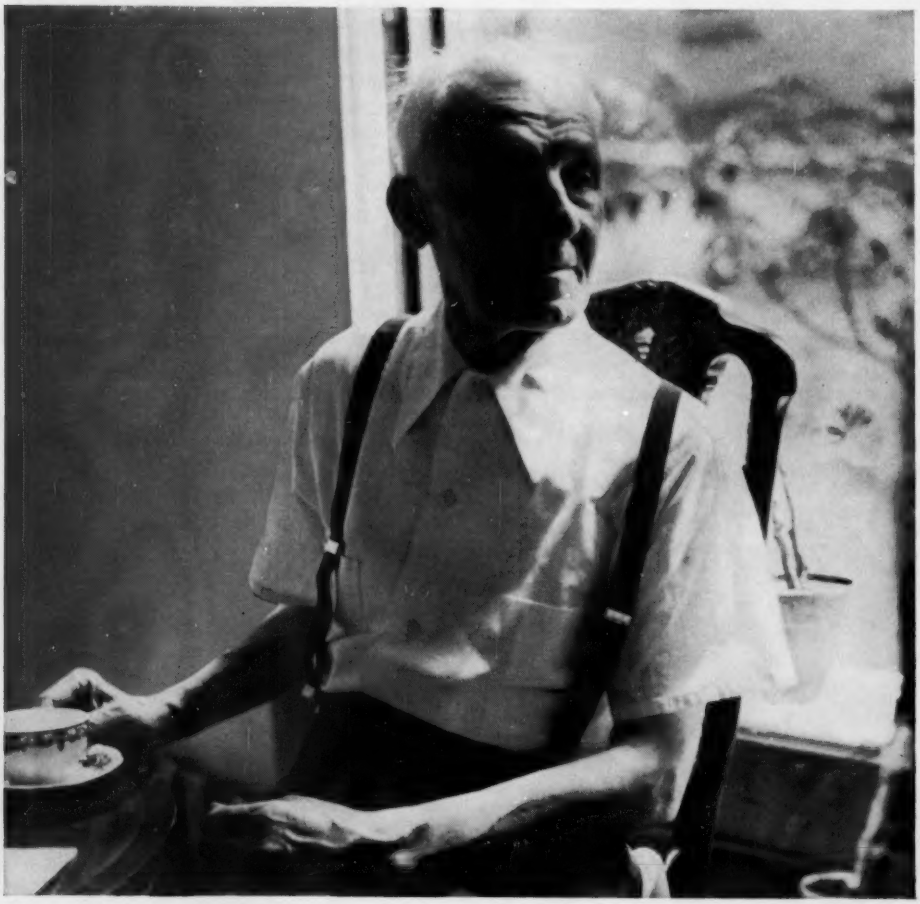

THE American Journal OF Gastroenterology

*A monthly journal of Gastroenterology, Proctology and Allied Subjects*
(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

VOLUME 28 JULY, 1957 NUMBER 1

# PANEL DISCUSSION ON DISEASES OF THE GALLBLADDER AND PANCREAS*

WILLIAM A. BARNES, B.A., M.D., *Moderator*†
MARY ANN PAYNE, B.A., M.A., Ph.D., M.D.‡
MICHAEL LAKE, A.B., M.D.¶
JOHN M. PEARCE, Ph.B., M.D.**
WILLIAM F. NICKEL, JR., M.D.††
and
JOHN A. EVANS, M.D.‡‡
New York, N. Y.

*Dr. William A. Barnes:*—It is hardly necessary to indicate to this group the importance of diseases of the biliary tract in the total care of our population, yet it is interesting to note that it has been estimated that 40 per cent of females over 50 years of age in this country are presumed to have gallstones and that biliary tract disease is the condition most commonly requiring surgery in patients over 70 years of age.

With the increasing age of our population, greater numbers of patients with neoplasms of the biliary tract and pancreas add to an already difficult problem.

To discuss these and various other aspects of diseases of the biliary system and pancreas, we have drawn upon the members of the Cornell University Medical College-New York Hospital staff. I should like to introduce them.

---

*Presented before the Third Annual Convention of the American College of Gastroenterology, New York, N. Y., 15, 16, 17 October 1956.

†Associate Professor of Clinical Surgery, Cornell University Medical College.
‡Assistant Professor of Clinical Medicine, Cornell University Medical College.
¶Assistant Professor of Clinical Medicine, Cornell University Medical College.
**Professor of Pathology in Surgery, Cornell University Medical College.
††Associate Professor of Clinical Surgery, Cornell University Medical College.
‡‡Professor and Chairman, Department of Radiology, The New York Hospital-Cornell Medical Center.

On the extreme left is Dr. Mary Ann Payne, Assistant Professor of Clinical Medicine, Cornell University Medical College, and Assistant Attending Physician, New York Hospital.

On my right is Dr. Michael Lake, Assistant Professor of Clinical Medicine, Cornell University Medical College, and Assistant Attending Physician, New York Hospital.

Then we have Dr. John M. Pearce, who is Professor of Pathology in Surgery, Cornell University Medical College, and Attending Pathologist, New York Hospital.

Next is Dr. William F. Nickel, Jr., Associate Professor of Clinical Surgery, Cornell University Medical College, and Attending Surgeon, New York Hospital.

Then we have Dr. John A. Evans, Professor and Chairman, Department of Radiology, the New York Hospital-Cornell Medical Center.

I think we must realize that the diagnosis of diseases of the biliary tract is not always readily made, and we should like to begin the discussion by briefly indicating what those conditions are that most closely resemble or simulate biliary tract disease. I will ask Dr. Payne if she will initiate the discussion.

*Dr. Mary Ann Payne:*—Dr. Barnes, as you stated initially, the diagnosis of biliary tract disease is often quite difficult, as symptoms suggestive of lesions in the biliary tract can be simulated by many other conditions. Practically any intraabdominal inflammatory process can be confused with acute or chronic cholecystitis, pancreatitis, duodenal ulcer and, of course, acute appendicitis. Less frequently, hiatus hernia, lesions of the hepatic flexure and even kidney lesions present problems in differential diagnosis.

In addition to these intraabdominal conditions, the internist and the surgeon are occasionally called upon to distinguish between acute cholecystitis and coronary artery disease. This is a favorite question on Surgical Boards. It can be a troublesome distinction in an acutely ill patient.

*Dr. Barnes:*—Now, when a patient has gallstones, we usually know it, but they don't always cause symptoms. Dr. Pearce, I should like you to tell us, if you can, how often do they cause symptoms?

*Dr. John M. Pearce:*—I would say approximately 50 per cent of them cause symptoms and 50 per cent are quite free of symptoms. Symptoms, of course, are more common in some groups of people, much more common in diabetics, and much less common in people with pernicious anemia, but in diabetics symptoms are not any more likely to occur than in those who are not diabetic.

*Dr. Barnes:*—It seems generally accepted that when gallstones cause symptoms, the gallbladder should be removed, but I should like to ask the members of the panel, how about the so-called silent stones? Should they be removed, and under what conditions should they not be removed?

Dr. Lake, what are your ideas on the subject?

*Dr. Michael Lake:*—I think it becomes a question of judgment in the individual case. If we have a woman of 70 and the stone is causing no particular trouble, I would leave it alone. If we come across a stone in a woman of 30, I would advise its removal.

I think in probing the history, you sometimes elicit symptoms which are not severe enough to warrant the consulting physician to advise removal. It is just a question of possible cancer of the gallbladder arising in connection with stones.

*Dr. Barnes:*—We have to take into consideration morbidity. Do you think that outweighs other considerations of the gallbladder in the various groups?

*Dr. Lake:*—I should say it does.

*Dr. Barnes:*—What is the incidence of carcinoma of the gallbladder?

*Dr. Pearce:*—In general, about 3 per cent of all fatal cases of cancer. It is about one-tenth as common as carcinoma of the stomach; however, when you get into the older age group, those you are talking about now, who have gallstones, the gallstones should be removed. In people over 60, the incidence of carcinoma of the gallbladder is much higher in those who have stones, about 12 per cent, so it seems to me that an elderly person otherwise in goood health, who is a good operative risk—and I do not mean very elderly—I mean someone approaching 60, in the 50's—it seems to me that if that person is a good operative risk the thing to do is take out the stone, even though it is causing no particular trouble, and get rid of the possible cause of carcinoma.

*Dr. Barnes:*—I should like to ask the surgeon, are you limited by any particular age at which you would say you should not remove the gallbladder, for stones?

*Dr. William F. Nickel, Jr.:*—I think the very presence of gallstones in the absence of definite contraindications to surgery, is an indication for cholecystectomy. A 70-year old patient, as you have stated in your opening remarks, is more likely to have symptoms from gallstones than from any other intra-abdominal disease. It is the most common surgical condition we encounter in old people, and I would advise cholecystectomy even in the elderly patient. The mortality rate for cholecystectomy in patients of 65 and over is a little less than 2 per cent as an elective procedure. It is a little less than 1 per cent in the patients under that age. I do not believe that the difference is great enough to warrant a statement that the older patient should not have the gallbladder out because the mortality rate is a little higher than in younger people.

*Dr. Barnes:*—Does anybody want to add anything further to the point?

Is there any difference in the possible complications in patients with a single large stone and those with multiple small stones, Dr. Nickel?

*Dr. Nickel:*—I don't know of any statistical difference, but there would seem to be a greater possibility of a patient with multiple small stones developing complications than the patient with one large gallstone, especially if the stone has remained silent for many years. Multiple small stones are much more likely to fall down into the common duct and produce symptoms of common duct obstruction. On the other hand, a large solitary gallstone can do things which would necessitate surgery too. The development of cancer for example is something everybody talks about nowadays. It is a very real complication of gallbladder disease whether single or multiple stones are present.

*Dr. Barnes:*—Dr. Evans, in what percentage of patients with cholelithiasis can the diagnosis be made by x-ray examination?

*Dr. John A. Evans:*—I think it is pretty generally recognized and admitted that cholecystography is perhaps one of the most accurate roentgenographic examinations that we have and the accuracy has been stated to range from 94 to about 97 per cent. Of course, that includes not only the general gallbladder calculi but also the inference of gallbladder disease by nonvisualization of a functioning gallbladder.

*Dr. Barnes:*—How do these figures that Dr. Evans has just given compare with the diagnosis of stones by duodenal drainage?

*Dr. Lake:*—I think the duodenal drainage can raise the percentage of positive findings where the x-ray is not diagnostic, or is negative, perhaps by an additional 4 or 5 per cent, but the figures Dr. Evans gave do not leave room for that. (Laughter)

In a series I reviewed some years ago, before the new Telepaque dye, the x-ray was correct in about 90 per cent. The biliary drainage gave positive findings in about 90 per cent, and one or the other gave positive findings in 97 per cent.

*Dr. Barnes:*—Why doesn't biliary drainage give it in 99 or 100 per cent?

*Dr. Lake:*—You may not get enough crystals appearing in the bile, although stones may be there.

*Dr. Barnes:*—What are the signs to look for?

*Dr. Lake:*—In the first place you see if you can obtain a "B" fraction, a concentrated bile, following stimulation with magnesium sulfate. This tells you if the gallbladder concentrates and empties. It serves as a check on the ability to visualize the gallbladder by x-ray, which of course depends in part on the cooperation of the patient in following instructions and on the ability of the liver to excrete the dye. If you get nonvisualization and a good "B" fraction, the x-ray should be repeated first by the usual technic and then if necessary by the intravenous technic.

If you don't get a dark bile, you may not be getting gallbladder contents, or you may be getting gallbladder contents from a gallbladder which does not concentrate. If you do not get gallbladder contents, you may not get any crystals, even if stones are present.

The most significant microscopic finding is the presence of cholesterin crystals.

*Dr. Barnes:*—So that you would say in the small percentage, of patients with stones who have a normal cholecystogram, that drainage may indicate there is a stone there?

*Dr. Lake:*—I would say that the finding of cholesterin crystals almost always means stones. Occasionally you find them in cholesterosis without stones. Calcium bilirubin masses are suggestive of stones, but not as diagnostic, as they may be present in stasis due to intrahepatic disease. Other significant findings are the presence of giardia, or biliary tract epithelium and bile-stained pus.

*Dr. Barnes:*—If in the examination of the patient you found no stones, but some Rokitansky-Aschoff sinuses, would you say that would be sufficient indication to subject the patient to cholecystectomy?

*Dr. Evans:*—Although the demonstration of Rokitansky-Aschoff sinuses is a common pathologic finding, it has been an uncommon one on x-ray studies. Up until recently, just as recently as 1955, only 11 cases of roentgen demonstration of Rokitansky-Aschoff sinuses were reported in the literature. To that number we added two cases from our own experience. Of these 13 cases, six showed a constriction of the proximal portion of the gallbladder, four were associated with calculi and nine of them were in women.

It is felt that Rokitansky-Aschoff sinuses are related to inflammation and increased pressure within the gallbladder and hence their radiographic demonstration is an indication for cholecystectomy. In one of our patients, however, the pathological examination of the gallbladder revealed no evidence of inflammation and there were no stones, so I think in that one case, at least, there is a reason to question the previously accepted dictum that the gross demonstration of Rokitansky-Aschoff sinuses is an indication for cholecystectomy.

I think in the future we will radiographically demonstrate these sinuses much more frequently, due to the improvements in the contrast media.

*Dr. Barnes:*—Assuming, then, that this patient is ready for operation, Dr. Payne, would you say there are any special studies on a patient in the category of biliary tract disease that one would want to have done before an operation?

*Dr. Payne:*—Elective surgery on the biliary tract is actually no more hazardous than any other type of comparable operative procedure. I don't know of any special studies that would be necessary once the diagnosis had been established.

*Dr. Barnes:*—Do you have to do any special liver function studies in a non-jaundiced patient in the middle-aged group who has gallbladder disease?

*Dr. Payne:*—Most patients with long-standing cholelithiasis do exhibit some degree of parenchymal liver disease. This can best be evaluated by the amount of bromsulfalein rentention present before operation. Frequently the values are in the neighborhood of 10-20 per cent of dye retained after a 45-minute test. This value usually returns to normal after removal of the diseased gallbladder. In cases of more severe liver damage, the cephalin flocculation and the serum bilirubin are helpful tests.

If cholesterol stones are suspected, a serum cholesterol is desirable, and one searches for other evidences of high cholesterol in the body. Since cholesterol elevation and stones frequently occur in hypothyroidism, a check on thyroid function is important. Pigment stones are usually associated with hemolytic anemia, so that careful evaluation of the hematopoetic system is in order in such cases.

*Dr. Barnes:*—One other point—I want to get back to Dr. Evans.

The next question is: Do you attach any particular significance to duodenal pneumatosis?

An air bubble was seen in the duodenal bulb in 130, or 10 per cent, of 1,300 cases of gastrointestinal series by Tapiovaara and Heinivaara. In two-thirds of the cases this was associated with a pathologic condition of the gallbladder. It is considered that gallbladder diseases produce disturbances in duodenal motility by adhesions or reflexes, and so account for the duodenal air bubble. Does that have any significance?

*Dr. Evans:*—I attach no significance to it. It is, of course, very common to see air in the duodenal bulb during gastrointestinal studies. I think it has nothing to do with the disease of contiguous structures, but whether or not one sees air in the duodenal bulb, I think depends largely on the anatomical development or type of duodenal bulb you have.

*Dr. Barnes:*—I should like to turn for a moment to consideration of the acute gallbladder. Dr. Pearce, as a pathologist, do you think one can tell from the physical examination and the laboratory data what pathological changes are going on in an acutely inflamed gallbladder?

*Dr. Pearce:*—Of course, we are always astounded by the lack of correlation between the clinical examination and laboratory examination, but I think in general there is a correlation. It is striking when a person with very few symptoms, not much fever and not much leucocytosis, is found at operation to have an almost gangrenous gallbladder. Many times the laboratory data does not help, but, just the same, if a person has had a previously nonpalpable gallbladder, and in a few hours it becomes tender; the white count goes up; he has

fever and a lot of pain, the chances are then that the best bet, in spite of lack of correlation, is that something is wrong with the cystic duct; the gallbladder cannot drain out, and he is developing empyemia and gangrene.

*Dr. Barnes:*—How often is jaundice present in such a state, and what causes that jaundice?

*Dr. Pearce:*—Jaundice is present in about 15 to 20 per cent of cases, and though it is partly speculation and cannot always be proven at the time of operation, the idea is that jaundice is caused by the cholangitis, always with acute cholecystitis.

There is an inflammation of the large duct, the common duct, and this often produces edema of the common duct, which occludes and prevents bile coming through. What helps prevent it is that the acutely inflamed gallbladder is not contributing, so there is edema preventing its coming through. On the other hand, there is less dynamic force behind it, which is another thing. Small stones may get in the common duct and cut it off, or even thick pus or mucus seals the epithelium in the gallbladder and will occasionally clog the common duct and, rarely, a group of enlarged lymph nodes near the ducts will press upon them.

I think the force of the probe is greater than the force of the body, which prevents the bile coming through, but not the probe.

*Dr. Barnes:*—What is your concept of the acute gallbladder as a surgical emergency? Is it an emergency for the surgeon, or should the patient be watched? What is your present thinking of the mode of handling a patient such as Dr. Pearce described with elevated fever, high white count, and signs and symptoms of acute cholecystitis?

*Dr. Nickel:*—It is my present feeling that acute cholecystitis is an emergency. It is just as much an emergency in a patient over 65 as in a patient under 65, perhaps more so. One has to take into consideration very definitely the patient's age when dealing with acute cholecystitis because the mortality is so much higher in the older age group whether or not surgery is employed.

So many complications can occur in the presence of acute cholecystitis that one must carefully weigh the consequences of conservative versus surgical management.

In the older age group one has to take into consideration, in addition to the patient's age, the duration of the disease. A younger patient can tolerate the presence of acute cholecystitis for a longer period than the older-aged patient can. The mortality rate in the older-aged patient with acute cholecystitis rises quite markedly the longer the condition exists. As the time interval increases, from the onset of the disease to the time he is operated upon, the mortality rate also increases.

I don't think there is any question that in the younger age group the treatment of acute cholecystitis as a surgical emergency is the best treatment, because the mortality rate is low, and it prevents any further attacks. Where the greatest judgment and care must be exercised is in the older age group.

*Dr. Barnes:*—Some of the patients you speak of hesitate about operation. Is there ever a place for cholecystostomy rather than cholecystectomy, which I assume you would do by choice?

*Dr. Nickel:*—Yes, there is a place for cholecystostomy. I don't think there is much place for it in younger patients but there is a place for it in the older age group with acute cholecystitis.

*Dr. Barnes:*—If cholecystostomy is done, is cholecystectomy indicated at a later stage, or should a patient feel, once over this attack, he can get along without any trouble in the future?

*Dr. Nickel:*—If the patient's life expectancy is less than two years, I don't see much point in subjecting him to an elective cholecystectomy. If he has some disease like cancer, or leukemia, which obviously is going to take his life within a short time, then I do not believe he should be subjected to cholecystectomy. In our experience at New York Hospital, with a group of patients, over the age of 55 who had cholecystostomy for acute cholecystitis, over 50 per cent of them have had complications from their disease later on. Over 50 per cent of them developed either another attack of cholecystitis, or they had a stone in the common duct. So it is my feeling at the present time that when a patient in the older age group has a cholecystostomy, one certainly should plan to perform a cholecystectomy at a later date as an elective procedure providing the life expectancy is greater than two years. The mortality in our hands has been less than 2 per cent in patients over 65 when the operation is performed as an elective procedure.

*Dr. Barnes:*—I should like to move for a few moments to another aspect of the topic. For many, many years, back into the 1870's, I believe, there was a similarity noted between the symptoms of gallbladder disease and coronary artery disease. Do these two diseases co-exist in a higher proportion, than by chance, in the older age group, Dr. Pearce?

*Dr. Pearce:*—Yes, they very definitely do. Gallbladder disease is quite as common in people with coronary artery disease as it is in the general population; also, such degenerative diseases of the arteries, aorta, and all the large atheroscleroses are much commoner and more severe in people with gallbladder disease than in those who do not have it. I suppose that may have something to do with, or may be caused, let us say, by the fact that both these groups of patients at some time in their lives, perhaps not when gallstones were first found, but at some time have had a high blood cholesterol.

*Dr. Barnes:*—It has been suggested that reflexes originating in the gallbladder may give rise to symptoms of cardiac disease, particularly of pain. What is supposed to be the mechanism of that?

*Dr. Pearce:*—I think the mechanism of that rises from the fact that the heart and gallbladder are innervated through the vagus nerve, and when the gallbladder is inflamed, the celiac axis reflex, gets into play, it decreases the tone of the vagus, and the decrease in vagus tone in turn decreases the force of contraction of the heart and the volume output of the heart, and signs and symptoms, of course, occur.

*Dr. Barnes:*—Dr. Payne, some improvements have been noticed in patients with anginal symptoms, and in the electrocardiogram after cholecystectomy for cholelithiasis. Do you care to comment on this?

*Dr. Payne:*—Back in 1942, Dr. Ravdin reported on a series of patients who had striking improvement of their anginal symptoms after removal of their gallstones. In several of these patients, before the tube was removed from the common duct, the original anginal symptoms were induced by inflation of the common duct. This was a very convincing demonstration of the relationship between increased pressure in the biliary tree and the anginal symptoms of which the patient complained.

A great number of animal experiments have been done to show the effect of biliary tract changes on the electrocardiogram. In dogs, changes in the electrocardiogram can be produced consistently by manipulation of the biliary tract, provided some previous injury has already been inflicted on the coronary circulation. One may deduce from this that the older patients whose coronary blood flow has already been compromised by arteriosclerotic changes might be sensitive to changes in the biliary tract. In this situation, the gallbladder disease may well trigger off the anginal symptoms.

There is the famous story of John Hunter, who is said to have died at a medical board meeting in the presence of his physician, Edward Jenner. Hunter had complained of attacks of pain, which he attributed to coronary insufficiency. Jenner had always insisted that Hunter had gallbladder disease. At autopsy, Jenner's clinical diagnosis was vindicated, as Hunter had a gallbladder full of stones. His death, however, was due to a coronary occlusion.

*Dr. Barnes:*—Do you think there is any evidence that coronary artery disease may be benefited by cholecystectomy?

*Dr. Payne:*—If the anginal symptoms are triggered off by a diseased gallbladder, there is no doubt that the symptoms will subside after a cholecystectomy. A respectable number of case reports substantiate this supposition. In general, however, it is a dangerous premise to consider extensive surgery in the face of symptomatic evidence of coronary insufficiency. Except in very carefully evaluated instances, this approach is not to be recommended.

*Dr. Barnes:*—Do you think manipulation during a routine cholecystectomy is likely to produce a deleterious effect on the coronary circulation?

*Dr. Nickel:*—No, I don't think there is any good evidence that manipulation of the gallbladder at operation in itself produces any changes in the coronary circulation. I think the more important factors are the proper premedication of the patient and proper care during the operative procedure. I think those things can affect it, and the manipulation during the gallbladder procedure probably does not.

*Dr. Barnes:*—I should like to come back to Dr. Evans. We know of stones in the gallbladder and the Rokitansky-Aschoff sinuses. What is the significance of polyps in the gallbladder?

*Dr. Evans:*—Somewhere along in the panel discussion someone is going to have to say, "I don't know," and I guess this is the time.

Polyps are difficult to be sure about radiographically. My degree of success and accuracy has been woefully small in that respect. There are certain technical measures that we go through with the hope that we can differentiate a polyp from a stone, or polyps from stones; however, these are not specific. Recently every time that I have thought I had a polyp, it has turned out to be a localized thickening in the wall of the gallbladder or a stone imbedded in the mucosa of the gallbladder.

I think that one always has to be suspicious of the possibility of a polyp when the lucid defect in the gallbladder remains in one position despite maneuvers to change its position or dislodge it.

As to what to do on a presumptive diagnosis, I will pass that to someone else.

*Dr. Barnes:*—The question was: what is the significance of polyps in the gallbladder? Dr. Nickel, do you want to continue?

*Dr. Nickel:*—I was thinking of polyps everywhere in the body, and we have rather changed our thinking about them. We used to think polyps in the rectum were rather harmless things, but I don't believe many of us do any longer, and we feel that when a polyp is present, it should be removed. In the case of the gallbladder that, of course, means cholecystectomy.

*Dr. Barnes:*—Dr. Evans, can you recognize frequently gallstones in the small intestine? In what percentage can you tell whether the stone causes obstruction? Do you see them frequently, or not?

*Dr. Evans:*—That is not a particularly frequent occurrence, and I think when it does occur, the clinical findings certainly are highly suggestive of this type of situation.

Now, with gallstone ileus we see that perhaps once a year, or once every two years. The gallstone usually traverses the small intestine without being impacted until it reaches the ileocecal valve, and then, of course, one gets the signs of small bowel mechanical obstruction. In addition, the tip-off to the diagnosis is usually evident by the demonstration of air in the bile ducts. The gallstone itself may not be visible on the abdominal scout film.

*Dr. Barnes:*—Moving down a little further in the biliary tree, from the gallbladder to the common duct, Dr. Nickel, would you say there is any clinical significance in the blood supply of the common duct? We don't hear very much about that. Is there anything one should know about the blood supply of the common duct that may have clinical significance in operative procedure involving that region?

*Dr. Nickel:*—There have been recent studies by Henley*, a British surgeon working in this country, which demonstrate that the blood supply to the common duct comes from two sources, one, the gastroduodenal artery, and the other the inferior pancreaticoduodenal artery. It is the gastroduodenal branch that supplies most of the blood to the common duct, and he showed that when it does not anastomose freely with the branch from the inferior pancreaticoduodenal artery, that if one ligates this vessel (gastroduodenal branch) proximally, it may result in gangrene of the distal end of the common duct. This may be a factor in producing some strictures of the common duct which cannot be explained otherwise.

*Dr. Barnes:*—In addition to the blood supply that may be important in diseases of the biliary tract, I should like to ask whether foci of infection in the biliary tract can be treated adequately by antibacterial agents alone.

*Dr. Payne:*—Antibacterial agents alone are not particularly effective in the treatment of biliary tract disease. As in other areas of the body, localized collections of pus must be drained and chronically infected areas with thick walls are difficult to reach with adequate concentrations of the drug.

Much has been said in the recent literature concerning the concentration of various antibiotics in the biliary tract. Chloramphenicol, for example, is concentrated 30 times, aureomycin, 16 times, penicillin 5 times and on down the list of new drugs. This may well be true in experimental studies, but the sad fact remains that the diseased liver and gallbladder fail to concentrate these agents as normal organs do. Therefore, in the situation where it is most needed, the mechanism is not operating.

*Dr. Barnes:*—Dr. Evans, what are the indications for intravenous cholangiography, and tell us a little bit about your experience with this diagnostic procedure.

*Henley: Brit. J. Surg. (July), 1955.

*Dr. Evans:*—To date at the New York Hospital we have done over 1,000 intravenous cholangiograms, without a fatality or serious complication. As a result of this experience we have come to regard skin testing as a futile, and conjunctival testing as a dangerous means of predicting whether or not a patient will have a systemic reaction. A negative test may create an unwarranted sense of security when, in fact, the patient may have a toxic reaction, and a positive test may deprive a patient who may not react at all, of a very essential examination.

Intravenous cholangiography, moreover, is a laborious and an expensive procedure, giving specific but at times limited results. If a patient is to receive the attention he deserves, we must recognize certain indications for the examination.

At the New York Hospital we recognize the following:

1. A patient with recurrent biliary tract symptoms after cholecystectomy, when jaundice is active or subsiding; and

2. Before cholecystectomy, with a poor risk patient, with a nonfunctioning gallbladder, and a common duct which the surgeon does not want to explore if he can avoid it; and

3. When there is a need to rule out gallbladder disease rapidly in a patient with acute abdominal symptoms; and

4. As a means of demonstrating the patency of a repaired or reconstructed common duct; and, finally,

5. As an occasional successful means of demonstrating calculi in a nonfunctioning gallbladder.

*Dr. Barnes:*—Do you have anything you would like to show on that? Do you want to show some pictures?

*Dr. Evans:*—Well, the first slide that I have relates to an attempt last year to shed some light on the causes of the postcholecystectomy syndrome. Here (slide) are analyzed the intravenous cholangiograms of 121 patients. These patients came in with pain and nausea, and intolerance of fatty food after cholecystectomy. In addition, a control study was made of 46 asymptomatic patients. In the asymptomatic group there were 121 patients, 100 women and 21 men. The common duct was inadequately visualized in 87 per cent of the group. The most common cause for nonvisualization was jaundice or hepatic insufficiency. In 26 patients symptoms could be explained wholly or in part by disease existing outside of the liver or the biliary tract, and these included duodenal ulcer, pancreatitis, hiatus hernia, and a few miscellaneous conditions.

As for the group of patients with pancreatitis, our findings were very inconsistent. Three patients showed a common duct of normal caliber. In two the

duct was dilated and in two not visualized. In none of these cases was there any characteristic tapering or displacement of the lower end of the common duct observed.

In 10 patients (8.3 per cent) there was an existing parenchymal disease of the liver, such as cirrhosis, hepatitis, cystic disease and tumor. Forty patients (33 per cent) showed abnormalities of the major bile duct, the most frequent abnormality being persistent cystic duct stumps. There were 20 patients (16.5 per cent) in this group. Nine patients showed strictures and adhesions.

There were 29 patients (24 per cent) in whom the only abnormality observed was dilatation of the common bile duct. Seven millimeters was arbitrarily set as the upper limit of normal common bile duct diameter. We could detect no constant relationship between the size of the common bile duct and the time that had elapsed since cholecystectomy.

In one patient the duct was not visualized in the presence of normal liver chemistries, and in 15 patients no abnormality whatsoever of the common duct was demonstrated.

Now, in the asymptomatic control group, there were 46 patients, 42 women and 4 men, from the follow-up surgical clinics of the New York Hospital who reported themselves free of any digestive complaints following cholecystectomy. In this group the common bile duct was adequately visualized in 44. Every type of duct abnormality that was shown in the symptomatic group was also demonstrated in the asymptomatic controls. In terms of percentages they were practically identical, 33 per cent in the symptomatic, and 34 per cent in the asymptomatic.

In the control group almost twice as many cystic duct remnants were observed as in the symptomatic group (28.3 as against 16.5 per cent). There were also two patients in whom calculi were demonstrated. In one the calculus was located at the junction of the left and right hepatic ducts, and in the other, the common bile duct was filled with faceted stones. In the control group, in two, the common duct was not visualized, and there were 8, or 17 per cent, with no abnormalities.

It is of interest that in both groups there was but slight difference in the time required for the contrast agent to reach the duodenum. In 72 per cent of the patients with postcholecystectomy symptoms, cholografin reached the duodenum in 30 minutes or less. In the asymptomatic controls, this figure was 53 per cent. While this is not a large group statistically, it does indicate that no conspicuous stasis of bile occurs in the recurrent biliary tract syndrome.

(Slide) This merely is a chart showing the percentage of patients and the diameter of the common ducts in both the postcholecystectomy symptomatic group, and the asymptomatic controls. You may see they practically overlap.

(Slide) This is an intravenous cholangiogram of a 53-year old woman, which shows a rather long cystic duct remnant. This patient had her gallbladder removed 15 years previously, and had had persistent upper abdominal pain and nausea after fatty foods since the operation.

(Slide) This intravenous cholangiogram shows a stricture of the common bile duct at a point where the cystic duct would normally enter. This patient had a cholecystectomy ten years prior to this.

(Slide) This is a 53-year old woman who entered the hospital with symptoms of coronary artery disease, had electrocardiographic findings to support this. Eight days after admission she developed nausea and abdominal pain. An intravenous cholangiogram was done and demonstrated this large stone which was surgically removed, and the patient has been free of complaint since.

(Slide) This is a 40-year old woman in the asymptomatic control group. She had no digestive complaints at all, and was merely asked to have an intravenous cholangiogram as part of the control group. As you see here, her common duct is packed with many stones, and, in addition, she has a large cystic duct.

(Slide) This is another intravenous cholangiogram which shows many stones in the common bile duct.

(Slide) Another intravenous cholangiogram. The tomogram on the right shows three clearly defined stones in the common duct which were difficult to see on the conventional film.

(Slide) This slide shows a stone in the cystic duct remnant.

(Slide) This is a tomogram of an intravenous cholangiogram, showing a cystic duct remnant.

*Dr. Barnes:*—Dr. Lake would like to ask a question.

*Dr. Lake:*—Did you visualize the pancreatic duct in any of these cases, particularly in the ones where you assumed there was spasm of the sphincter?

*Dr. Evans:*—In none of our cases have we demonstrated the pancreatic duct.

*Dr. Barnes:*—What do you think is the explanation for this so-called postcholecystectomy syndrome in the large group of patients in whom you determined there were no abnormalities?

*Dr. Evans:*—Well, that is a $64 question, to which I am again going to have to say, "I don't know."

*Dr. Barnes:*—Dr. Nickel, it has been stated frequently that after cholecystectomy and choledochostomy stones that reform in the common duct, or which

may be left in the common duct, may frequently be removed, not by reoperation, but by irrigating solutions and smooth muscle relaxants. What is your experience in this?

*Dr. Nickel:*—A certain number of stones will pass through the common duct whether left behind at operation or by dropping down into the common duct during the natural course of the disease. When a stone is left behind and makes its presence known by the usual signs, we are faced with a problem of what to do, and when to do it. One of the things that has been advocated by a number of surgeons is to try to dissolve the stone with various solutions, one of the solutions commonly used being ether. I have seen this used on several occasions and have tried it myself on several occasions without success. I hesitate to advocate the use of irrigating solutions until we find some more effective agents than we have at present.

So far as the use of relaxants is concerned, I would certainly be in favor of trying them. There is none that I know of that works very well, but certainly there is no objection to trying. Eventually, though, we have to decide, as to when to operate on these patients the second time and extract the stone surgically.

*Dr. Barnes:*—I should like to ask Dr. Payne if she will give us a few words briefly stating some of the studies that may be helpful in the differential diagnosis of jaundice. We are all familiar with the common ones, but what newer methods are there, and what of the transaminase determination in the differential diagnosis? When is that useful?

*Dr. Payne:*—The problem is actually made very complex because of the multitudes of tests which we use to study liver function. Since the different functions of the liver may vary independently of each other, we need a variety of indices to evaluate total damage. The great danger, of course, lies in misinterpretation of the tests.

It is useful to remember that the clinical diagnosis is primarily the most important one. The laboratory tests are designed to prove or disprove that presumptive diagnosis. In order to use tests efficiently, we must know what we are looking for.

The second principle in using laboratory tests efficiently is to remember that liver function tests fall into two broad patterns, those which test the function of hepatic, parenchymal cells, and those which test the excretory system, the biliary tract. Anyone who expects more than this from liver function tests is bound to be disappointed. The tests do *not* tell us the *cause* of the damage of the parenchymal cells, *nor* do they tell us the *site* or *cause* of the obstruction of the biliary radicles.

Low serum albumin, prolonged prothrombin, elevated cephalin flocculation and thymol turbidity tests and increased retention of bromsulfalein are all indications of poor function of the liver parenchyma. Obstruction to the biliary tract is associated with elevation of alkaline phosphatase and of cholesterol, and usually of direct bilirubin.

Transaminase (S.G.O.) serum glutamic oxaloacetic, is not in the true sense a test of liver function, but rather a measure of liver injury. This is a tissue enzyme released as cells become necrotic. Its high concentration in heart and liver tissue accounts for the striking use in the serum levels on injury to these organs. In coronary occlusion, serum levels of 200-300 units of transaminase are frequently observed. In hepatitis, however, levels in the order of 1,000, 2,000 and 3,000 units may occur. These levels are transitory, and occur early in the course of hepatitis. They may, therefore, easily be missed. When, however, serum transaminase levels of this magnitude are found, it may be considered strong positive evidence for the diagnosis of hepatitis. This test is very helpful in distinguishing hepatitis superimposed on pre-existing cirrhosis and in the diagnosis of hepatitis of the cholangiolitic variety. Here other tests of parenchymal damage may fail to become positive, and the elevated transaminase may be the only chemical aid in diagnosis.

*Dr. Barnes:*—Thank you, Dr. Payne.

Assuming we have gone through that and arrived at the point where we are going to operate on the patient, what would you say would be your indications, Dr. Nickel, for exploring the common duct?

*Dr. Nickel:*—Certainly the presence of jaundice or a history of jaundice, or when one has exposed the common duct and finds it dilated or thickened, or when we find on palpation that there is something in the common duct; or perhaps a stone will have been demonstrated by operative cholangiogram; or perhaps before operation by an intravenous choledochogram. The presence of multiple small stones in the gallbladder, simply because of the increased possibility of stones lodging in the common duct, is also considered to be an indication for exploration by some surgeons.

There are other less usual causes for exploring the common duct, which I won't go into now, but those are the main ones.

*Dr. Barnes:*—Unfortunately, one of the causes of operation upon the biliary tract for a second time is the damage wrought at the first operation, usually a simple cholecystectomy. In an attempt to control bleeding from the cystic artery, blind clamping may injure the common duct with immediate or subsequent obstruction to the flow of bile. Even adequate visualization of the normal anatomical relationships in the vicinity of Calot's triangle before applying clamps may not prevent similar complications as almost occurred in the following case.

(Slide) This case illustrates one of the important reasons for removing a gallbladder from "above downwards" and not from the cystic duct towards the fundus, as commonly done.

The patient, a 52-year old woman, had had symptoms of gallstones for several months. At operation a perfectly normal-appearing cystic duct was found entering what appeared to be a normal common duct in the usual position. For some reason we decided to explore a little higher rather than immediately clamping the cystic duct, and we found what you can see appeared to be a second cystic duct extending from the neck of the gallbladder to the junction of the right and left hepatic ducts. We then examined more closely the common hepatic duct and found that while bile was flowing freely through the second cystic (accessory bile) duct, no bile was apparent in this supposedly normal segment of common hepatic duct, and on aspirating it, no bile was obtained.

We incised the common hepatic duct and there was no lumen. Hence the only way this patient's bile passed from the liver to the intestine was along the accessory duct and down the cystic duct to the normal common bile duct. The gallbladder was removed down to the junction of the accessory duct and the normal cystic duct and a tube inserted at the junction. The patient drained but little bile after operation, and, as before, the only way bile could pass was through this accessory duct and cystic duct. If the cystic duct had been removed in a routine manner, it would have left a situation tantamount to ligation of the common duct.

We feel that removal of the gallbladder from "above downward" to the cystic duct rather than beginning at the cystic duct and working toward the fundus is an important factor in avoiding injury to vital structures.

(Slide) Here is the cholangiogram postoperatively, showing the absence of contrast media in the segment where the common hepatic duct normally should lie and the course of the dye in the accessory duct to the cystic duct through the common bile duct and into the duodenum.

*Dr. Barnes:*—Dr. Nickel, what is the preferred method of handling stricture of the common duct which may occur? What is the best method to restore continuity?

*Dr. Nickel:*—The best method to use is immediate end-to-end anastomosis at the original operation. If that is done, that group of patients gets the best long-term results.

*Dr. Barnes:*—If the strictured segment of the common duct is too long for end-to-end anastomosis—suppose you come back and find the strictured segment several centimeters too long, what procedure do you follow?

*Dr. Nickel:*—If your strictured segment is too long, then one has to improvise, and the best method that has been found to overcome long strictures

of the common duct is the so-called Roux-Y method, using a defunctionalized loop of small intestine approximately 30 cm. long, anastomosed to the stump of the common duct, and then restoration of continuity by an end-to-side anastomosis below.

*Dr. Barnes:*—We have many other questions concerning problems of the biliary tract, but I should like to spend the next few minutes on the consideration of pancreatic disease.

Dr. Nickel, what are the usual clinical manifestations of annular pancreas?

*Dr. Nickel:*—One of the most common, and perhaps the outstanding manifestation of annular pancreas, is nausea and vomiting. The x-ray signs of annular pancreas are a defect in the second portion of the duodenum, and obstruction at that point due to the annular pancreas. In the older patients pain may be one of the outstanding symptoms and in infants jaundice occasionally occurs depending on the point where the obstruction lies.

*Dr. Barnes:*—Does the x-ray man usually make an unequivocal diagnosis of annular pancreas? What other conditions must be considered in the differential diagnosis?

*Dr. Evans:*—I don't think the radiologist can make an unequivocal diagnosis but he should be able to offer a presumptive diagnosis. At the very least, he should suggest the diagnosis. There are other conditions that enter into the differential diagnosis.

Dr. Nickel has stated that annular pancreas almost always occurs in the second duodenum. It occasionally is seen in the third duodenum, and may occasionally be seen in the first duodenum.

The radiographic appearances are two. Perhaps the most common is a concentric narrowing of a segment of the descending duodenum. The other is an eccentric indentation, usually on the right lateral aspect of the duodenum. On a preliminary scout film of the abdomen before using barium, the dilated stomach and duodenal bulb produce the so-called "double bubble effect".

The other conditions which one might consider in differential diagnosis are duodenal atresia and congenital duodenal band, and occasionally the rare condition of a duodenal volvulus.

*Dr. Barnes:*—Dr. Pearce, would you say there are any anatomical considerations that would dictate the proper management of annular pancreas?

*Dr. Pearce:*—I think there certainly are, since the common bile duct and pancreatic duct, going to the ampulla of Vater, and the splenic artery goes through this annular pancreas, one must be careful not to interfere. In many instances I would prefer not to cut the pancreatic ring at all, but anastomose the jejunum to the stomach.

*Dr. Barnes:*—Are there any anatomical considerations that would dictate the proper management of annular pancreas, Dr. Nickel?

*Dr. Nickel:*—Duodenojejunostomy certainly is the preferred method, rather than a gastrojejunostomy. The latter fails to properly drain the first portion of the duodenum.

*Dr. Evans:*—May I just inject a comment here? I think perhaps the duodenostomy and jejunostomy sidetracking procedures are preferred because of the fear of injuring the common bile duct or main pancreatic duct, and also, many of these cases of tight annular pancreas have an associated atresia. Even though the band is removed, the duodenal atresia may not be recognized at the time and obstruction persists.

*Dr. Barnes:*—One of the most baffling diseases and one that causes both medical people and surgeons many sleepless nights, is pancreatitis. Dr. Lake, will you tell us a little about the possible etiologic factors in the development of this disease?

*Dr. Lake:*—Dr. Barnes, we still do not know all the answers, although we all know that about 50 per cent of these are associated with biliary tract disease, and we know that perhaps a third more are associated with chronic alcoholism. Probably 75 or 80 per cent are associated with one or the other, but we still have a fair number of cases where the etiology is not apparent, and even in those associated with biliary tract disease, correcting the disease does not always cure the pancreatitis.

The mechanism by which pancreatitis is caused in the presence of biliary tract disease is a very controversial matter. You all know Opie's common channel theory, where a stone was present at the junction of the two ducts, and bile was diverted into the pancreas; however, in less than 5 per cent of the cases is this found at operation. Therefore, many people have assumed that a spasm of the sphincter of Oddi serves the same purpose and that the entrance of bile into the pancreatic duct causes pancreatitis. I seriously question whether that is true, and the value of treatment based on this theory, such as sphincterotomy, I think, has not yet been established.

I should like to hear the surgeon's comment on that.

In addition there are a few cases that are infectious, such as we get in mumps. There is experimental evidence that deficiency of amino acids, particularly methionine, can cause pancreatitis. Obstruction is certainly a factor in many cases, be it in the ampulla or within the ducts of the pancreas.

Dr. Nickel has reported a case I recall that had an ampullary carcinoma, where repeated attacks of pancreatitis occurred, which were relieved by removing the obstruction.

Dr. Porter, at Columbia University, told me of a case that had a tumor in the midportion of the pancreas, obstructing the duct, and distal to the tumor was clearcut pancreatitis, and proximal to the tumor the pancreas appeared normal. Whether obstruction alone could cause pancreatitis, or additional factors which activate the enzymes are necessary, I do not know.

*Dr. Barnes:*—Dr. Pearce, what is the incidence of biliary tract disease in patients with acute pancreatitis?

*Dr. Pearce:*—It is very, very common, somewhere around 70 per cent of patients with pancreatitis have biliary tract disease, and about 50 per cent of them have stones. The two are closely associated.

*Dr. Barnes:*—Dr. Evans, is the common channel theory of acute pancreatitis borne out in your studies with Cholografin? That is, has it shown the pancreatic duct outlined in a high percentage of such cases? According to what Dr. Lake said before, can this help us at all in making any inroads into the pathogenesis?

*Dr. Evans:*—In our experience with the intravenous methods, we have never actually demonstrated reflux or entrance of opaque media in the pancreatic duct.

*Dr. Barnes:*—In a case with acute pancreatitis you sometimes see a dilated loop. What is this so-called "sentinel" loop of acute pancreatitis supposed to be?

*Dr. Evans:*—I think it merely reflects the peritoneal insult that accompanies pancreatitis, as you might expect it to reflect peritoneal insult of many other conditions.

*Dr. Barnes:*—Dr. Lake, why does tetany sometimes occur in these cases?

*Dr. Lake:*—It is simply due to a fall in blood calcium. The calcium is depressed by its fixation by the fatty acids in areas of fat necrosis.

*Dr. Barnes:*—How about the gastrointestinal bleeding frequently or occasionally seen in these patients, Dr. Nickel?

*Dr. Nickel:*—Well, that is probably due to the gastritis or duodenitis that accompanies pancreatitis in some cases.

*Dr. Barnes:*—How about the medical management of acute pancreatitis, Dr. Lake?

*Dr. Lake:*—It is not very successful. If there is alcoholism, you have to put—

*Dr. Barnes* (Interposing):—"Acute!"

*Dr. Lake:*—Acute pancreatitis, I believe, is chiefly a medical problem so far as treatment goes, in the absence of certain complications. We usually try to keep the stomach empty by using suction. We replace fluids and electrolytes as necessary. We try to decrease vagal stimulation on the theory that it will

decrease the formation of enzymes, by using blocking agents such as Pro-Banthine or Banthine. We use Demerol in preference to morphine for pain, because morphine causes spasm at the end of the pancreatic duct. We replace calcium and other electrolytes, if necessary, and we try to sit tight on these cases. Some of them develop abscesses or other complications in the abdomen. Some of the cases have a common duct stone which needs to be removed. Acute cholecystitis may develop.

We use antibiotics as a prophylactic measure because the mortality has been shown to be lower when antibiotics are used than when they are not used.

*Dr. Barnes:*—Someone in the audience wants to know what percentage of acute cholecystitis cases are accompanied by an associated acute pancreatitis, and, is the presence of acute pancreatitis with the acute gallbladder a contra-indication for prompt cholecystectomy.

*Dr. Nickel:*—Approximately 50 per cent of patients with acute pancreatitis have biliary tract disease. The question, I believe, is, what percentage of patients with acute cholecystitis also have acute pancreatitis. Is that right?

*Dr. Barnes:*—Yes.

*Dr. Nickel:*—I do not know that I can answer that. I do know that many patients with acute cholecystitis also have acute pancreatitis, but I do not think that the presence of acute pancreatitis, unless symptoms of shock are present, is a contraindication to cholecystectomy. Those patients who have acute pancreatitis and associated biliary tract disease do very well following removal of the gallbladder, so that I do not think that the presence of the acute pancreatitis, associated with acute cholecystitis, is a contraindication to removal of the acute gallbladder.

*Dr. Barnes:*—Dr. Nickel, you also are asked to comment on your experience with and estimation of the usefulness of cholangiography at the operating table.

*Dr. Nickel:*—The use of operative cholangiograms and indications for them are approximately the same as for exploration of the common duct. The two go hand in hand. If we believe that an operative cholangiogram is indicated, we also explore the common duct, and vice versa.

*Dr. Barnes:*—What is your feeling about the use of biliary tract drainage in cases of known hepatitis which do not clear under conservative management?

*Dr. Nickel:*—I have not had any experience with drainage of the biliary tract in the presence of hepatitis. I would certainly try to avoid operating on a patient if I knew the patient had hepatitis, although I am aware that there is a difference of opinion in that field, and there are some surgeons who advocate drainage of the biliary tract in certain cases of hepatitis. I have had no experience

with it and I would hesitate to operate on a patient if I were sure the patient had hepatitis.

*Dr. Barnes:*—Dr. Evans, does pigment stone have any different shading radiologically?

*Dr. Evans:*—Well, I think that I wouldn't say any tremendously helpful or significant features.

*Dr. Barnes:*—Above what level of jaundice, preferably in terms of serum bilirubin, is it impractical to attempt intravenous cholangiography, and how does this compare with the older oral or intravenous contrast media?

*Dr. Evans:*—I would say in our experience it is uncommon to obtain opacification of the common bile duct in patients whose serum bilirubin is above 3 mg. per cent. We do obtain opacification of the common duct in patients who are clinically jaundiced, but the clinical jaundice frequently lags behind the level of the serum bilirubin. When the bilirubin level is between 2.5 and 3 mg. per cent, it is uncommon to get adequate visualization of the extrahepatic duct system.

I would say that the intravenous method demonstrates the extrahepatic ducts in a high percentage of cases and that there is better opportunity of demonstrating them than would be the case using the current oral media.

I may look it, but my experience does not permit me to comment on the comparison with iodophenolpthalein. (Laughter)

*Dr. Barnes:*—Also, Dr. Evans, what is your opinion of the normally outlined but noncontracting (after a fatty meal) gallbladder?

*Dr. Evans:*—I place no significance on the contractability of the gallbladder following fatty meals. Fatty meals vary tremendously in their effectiveness on the gallbladder. I think that it is unwise to draw any conclusion from a lack of contraction of the gallbladder following a fatty meal.

*Dr. Barnes:*—Dr. Nickel, what are the indications for the various surgical operations in chronic pancreatitis?

*Dr. Nickel:*—That is the sixty-four-*thousand*-dollar question!

Surgeons are at a loss to deal with these patients, and many operations and treatments have been devised. One thing I think everyone is agreed upon, though, and that is that disease of the biliary tract in the presence of pancreatitis is in itself an indication for surgery. Patients who have gallstones with pancreatitis do well following removal of the gallbladder. That leaves about 50 per cent who do not have associated biliary tract disease. A number of operations have been devised to deal with this group. One of the most popular ones

has been sphincterotomy designed to overcome the factor of obstruction at the sphincter of Oddi, and regurgitation of bile into the pancreatic duct. There is a great difference of opinion as to whether regurgitation of bile into the pancreas can produce pancreatitis. There is some agreement, however, that an obstructive factor is present in a definite percentage of these patients with pancreatitis who do not have associated biliary tract disease. The rare case of obstruction due to stone in the ampulla of Vater and obstruction due to tumors has been noted.

Other operations have been devised, such as transplantation of the common duct or of the pancreatic duct so there would no longer be a common channel, and also to overcome the obstructive factor.

A recent operation devised by Duval is a retrograde anastomosis. The tail of the pancreas is implanted into the jejunum when obstruction can be demonstrated at the sphincter of Oddi, and in this manner the surgeon hopes to overcome the obstructive factor. Other operations, such as gastric resection and gastroenterostomy, have been advocated. Vagotomy has been advocated as the treatment for recurrent pancreatitis, the object being the diminution of pancreatic secretion. By the same reasoning, ligation of the pancreatic duct has been advocated, the rationale being that ligation of the duct would produce atrophy of the gland and would thus decrease pancreatic secretion.

All these various operations have been effective on occasion. They are not universally accepted, however, and until we know more about the etiology of the disease, I believe there will not be a standard operation except cholecystectomy and exploration of the common duct when indicated.

*Dr. Barnes:*—Thank you, Dr. Nickel.

Dr. Payne, how does thorazine affect the liver cells?

*Dr. Payne:*—The exact mechanism by which thorazine causes jaundice is not known. Early in the course of thorazine hepatitis the changes seen on biopsy of the liver are indeed minimal. The liver cells themselves do not show any change. There may be a moderate number of inflammatory cells around the portal areas. The injury apparently occurs in the cholangioles of the biliary tree. These cells are probably responsible for the elevation of alkaline phosphatase, which is a consistent early finding in thorazine hepatitis. There are a number of observations to suggest that the effects of thorazine are due to an allergic response—an idiosyncrasy of certain individuals to this drug. The occurrence of jaundice does not seem to be related to the size of the dose; the response comes on ten days to two weeks after ingestion of the drug and lastly, a significant eosinophilia frequently accompanies this type of hepatitis.

*Dr. Barnes:*—I have a question for Dr. Evans.

When preliminary films of the gallbladder show no stones, are delayed films of value in the diagnosis of cholelithiasis?

*Dr. Evans:*—This is in a cholecystogram?

*Dr. Barnes:*—Yes.

*Dr. Evans:*—I would say that delayed films in my experience have not been of any merit or value, when using the oral media. There is some advantage when using intravenous media to extend the duration of the examination, even though the gallbladder is not visualized at the normal time.

*Dr. Barnes:*—Dr. Payne, is Hanot's biliary cirrhosis (the classical description of this disease) the same as chronic postnecrotic cirrhosis?

*Dr. Payne:*—Because of the lack of sufficiently precise criteria, the terms Hanot's cirrhosis or hypertrophic biliary cirrhosis came to be applied to a variety of clinical syndromes. It would appear that many of these conditions produce the same type of histological changes in and around the small cholangioles of the liver. Thus, thorazine hepatitis, the toxic reaction of arsephenamine and Mapharsen and methyltestosterone, and some of the cases of viral hepatitis would cause similar changes. The final histological picture following viral hepatitis is varied, however, and may be a postnecrotic-type cirrhosis.

*Dr. Barnes:*—Dr. Lake, does section of the sphincter of Oddi effect dysfunction of the gallbladder?

*Dr. Lake:*—I would think so. Bile normally flows from the liver to the sphincter of Oddi. If the sphincter is closed, as it normally is between meals, it backs up into the gallbladder and is concentrated there. The gallbladder empties after a fatty or protein meal. If the sphincter has been cut, the gallbladder will not fill.

*Dr. Barnes:*—Here is a lengthy one: suppose you had an acute gallbladder, with the common duct dilated, with acute inflammation of omentum around the common duct, and you were to take out the gallbladder, would you explore the common duct at this time and put in a T-tube? If you also found at that time that the patient had a perforated duodenal ulcer, which did not show on x-rays, what would you do in this case? (Laughter)

*Dr. Nickel:*—I hope I never run into that! I don't know what I would do. Closure of the ulcer is one thing that I would do. Whether I would explore the common duct would be another question.

*Dr. Barnes:*—This is another long one and somewhat difficult to read. I will summarize it.

Dr. Payne, this patient, 50 years old, or younger, had a coronary occlusion, and work-up right afterwards showed a gallstone or gallstones, with severe

pain that required morphine with each attack, and also duodenal and gastric ulcers. (Laughter) What would you do first?

*Dr. Payne:*—This is the type of problem where one plays for time. If biliary colic persists in the face of a recent coronary occlusion, one might consider a cholecystostomy to decompress the biliary tract. After the coronary has healed and the other conditions stabilized, one could then do an elective cholecystectomy.

*Dr. Barnes:*—One more question, and it is to Dr. Nickel: in what condition would you advise splanchnicectomy for pancreatitis?

*Dr. Nickel:*—I think the only time it has a place is in intractable pain due to chronic pancreatitis or pancreaticolithiasis.

# CHYLOTHORAX AND CHYLOUS ASCITES
## A STUDY OF 302 SELECTED CASES*

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Chylothorax and chylous ascites are not disease entities, merely fascinating sequelae of an internal lymph fistula of obstructive or traumatic origin. This review has been prompted by the desire to ascertain the value of recent advances in lymph physiology in the diagnosis and treatment of these conditions.

Lymph nomenclature has been confused by the use of standard terms in different senses by various investigators. To avoid misinterpretation, it is necessary to specify the meaning certain key words carry in this report. "Pure lymph" designates origin from a specific tissue or viscus; by contrast, "mixed lymph" signifies a pooling of pure lymph from two or more sources. The adjective "chylous" rather than the noun "chyle" is used to describe lymph of a high fat content. With the exception of intestinal lymph, chyle is never a pure lymph from a single viscus; chyle is usually mixed lymph composed of lymphatic drainage from several viscera and regions of the body.

Rigid criteria were established for acceptance of cases from the literature. Of the 479 cases reviewed, 294 were found to meet our criteria of a chylous fluid plus one or more of the following:

1. Free fat microscopically
2. A fat content higher than plasma
3. A protein content half that of plasma
4. Surgical or autopsy evidence of lymphatic fistulae.

These 294 cases selected from the literature, combined with 8 cases previously unreported from Charity Hospital of Louisiana in New Orleans (Table I), makes a total of 302 cases upon which this report is based. This study is distinctive in that the same method of case selection and study was used for both chylothorax and chylous ascites.

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Of the 294 cases collected from the literature, 77 were chylothorax; 126 were chylous ascites and 32 were chylothorax and chylous ascites combined.

## Incidence

As many selflimiting, nonmalignant cases of chylothorax and chylous ascites are unrecognized, and many chylous effusions secondary to malignancy are not reported, the low incidence is probably more apparent than real. Furthermore, the incidence of chylothorax and chylous ascites is increasing with the perfection of cardiovascular surgery, aortic grafts, and hepatic and pancreatic resections. A general idea of the incidence of chylothorax and chylous ascites can be gained from the 11 Charity Hospital cases included in this report. These cases

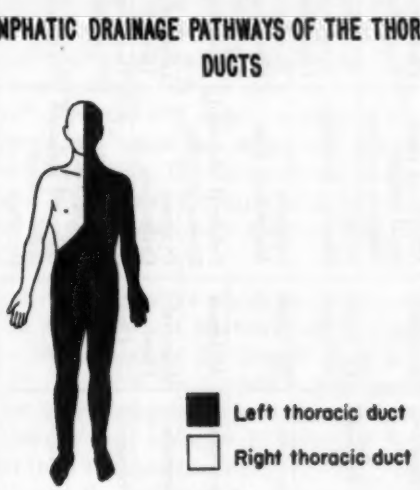


Fig. 1

were recognized between 1936 and 1956. A total of 1,168,760 general admissions were seen at that institution during this interval. No unusual distribution as to age or sex was apparent in the cases reviewed. As most of the reports made no mention of the color of the patient, no definite race distribution could be ascertained. In the 11 cases seen at Charity Hospital, however, 5 were colored.

## Anatomy

The *cisterna chyli* and thoracic duct are constant only in their variability. The *cisterna chyli* is first noted in the 23 mm. human embryo[58]. The beginning thoracic duct is seen to project upwards into the thorax and downward from the jugular bulb. In the 30 mm. embryo, the thoracic duct system exists as two distinct, bilaterally symmetrical channels with numerous cross anastomotic

TABLE I

CHYLOTHORAX AND CHYLOUS ASCITES CASE REPORTS DATA FROM 11 CASES ADMITTED FROM 1936-1956

| No. | Year | Age | Sex | Race | Etiology | Classification | Location | Symptoms | Treatment | Results | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | 1932 | 24 | M | C | Cirrhosis | Spontaneous | Rt. Chest Abdomen | Dyspnea shock Peritonitis | Thoracentesis Paracentesis | Died | Dilated lymphatics at autopsy |
| 2. | 1939 | 42 | F | C | Unknown | Spontaneous | Abdomen | Enlarged abdomen Pedal edema | Paracentesis Pneumo-peritoneum | Deserted | Free fat in fluid aspirated |
| 3. | 1940 | 40 | F | W | Hodgkin's | Spontaneous | Abdomen | Abdominal pain Pedal edema Bleeding tendency | Paracentesis Laparotomy | Died | Thoracic duct obstructed at autopsy |
| 4. | 1943 | 43 | F | W | Adeno CA of Ovary | Spontaneous | Lt. Chest Abdomen | Shock Bleeding tendency | Thoracentesis Paracentesis Laparotomy | Died | Thoracic duct obstructed at operation |
| 5. | 1944 | 14 | M | W | Crushing injury | Traumatic | Rt. Chest | Dyspnea Abdominal pain | Thoracentesis Phrenic crush Chyle infusion | Died | Thoracic duct narrowed at autopsy |
| 6. | 1951 | 6 mo. | F | W | Congenital | Spontaneous | Abdomen | Enlarged abdomen Edema of vulva | Paracentesis | Cured | High fat content of fluid aspirated |
| 7. | 1951 | 28 | F | W | Fall | Traumatic | Bilateral Chest | Dyspnea Orthropnea | Ligation of Thoracic duct | Recurred | Free fat in fluid aspirated |
| 8. | 1951 | 39 | F | C | Unknown | Spontaneous | Lt. Chest Abdomen | Dyspnea Pedal edema Weight loss | Thoracentesis Paracentesis | Unknown | High fat content of fluid aspirated |
| 9. | 1951 | 35 | F | W | Unknown | Spontaneous | Abdomen | Enlarged abdomen Pedal edema Diarrhea | Paracentesis | Cured | High fat content of fluid aspirated |
| 10. | 1925 | 2 mo. | M | C | Congenital | Spontaneous | Abdomen | Enlarged abdomen Hydrocele | Paracentesis Laparotomy Gelfoam application | Unknown | Leaking lymphatics seen at operation |
| 11. | 1952 | 56 | M | C | Lymphoma | Spontaneous | Lt. Chest Abdomen | Dyspnea Weight loss | Thoracentesis Paracentesis Laparotomy | Died | Thoracic duct obstructed at operation |

Cases 5, 6, and 9 were previously reported.

F: Female M: Male W: White C: Colored Lt.: Left Rt.: Right

vessels. One of these collateral channels, at the level of the fifth thoracic vertebra, dilates and the lower left part of the duct undergoes atrophy due to pressure from the aorta. Most of the upper right duct atrophies except for a small segment which forms the right thoracic duct. This leaves a single duct extending up the right side of the midline to the fourth or fifth thoracic vertebral level where it crosses obliquely to the left side[58]. This explains why low thoracic duct injuries give a right chylothorax and high thoracic duct injuries give a left chylothorax.

The *cisterna chyli* lies on the anterior surface of the second lumbar vertebra and receives lymph from the liver, intestinal, descending thoracic and lumbar trunks. From the *cisterna chyli*, the thoracic duct extends upwards through the aortic hiatus between the aorta and azygos vein. It then enters the superior mediastinum behind the aortic arch and subclavian artery between the left side of the esophagus and the left parietal pleura. Below the level of the eighth dorsal vertebra the duct is usually single (92 per cent) and this is the level of elective transthoracic ligation[162]. The thoracic duct is separated from the esophagus by a thick layer of adipose and connective tissue and is bound to the vertebral column by the prevertebral fascia. The thoracic duct is retropleural throughout its course in the thorax. This allows for chyle to collect retropleurally and produce clinically a case of chylothorax only after rupture through the pleura, at the lateral pleural ligament[133].

The thoracic duct contains valves which vary in position and number except the one at the lymphaticovenous junction[58] which guards against the reflux of blood into the lymphatic tree. As the thoracic duct ascends into the neck, it arches as high as 5.5 cm. above the clavicle to join the venous system[58]. The thoracic duct joins the internal jugular vein as a single trunk in 47 per cent of the cases. The left thoracic duct averages 18 inches in length and in addition to the lymph received from the *cysterna chyli*, receives lymphatic drainage from the left side of the thorax, the left lung, the left upper extremity, and the left side of the head and neck. Histologically the thoracic duct is from 4 to 6 mm. in diameter and is composed of endothelial, muscular and connective tissue[162]. Acrevedo[1] from his studies on cats has shown the duct to be a contractile organ which is under the control of both the sympathetic and parasympathetic system.

The right lymphatic duct averages only one inch in length and drains the upper surface of the right lobe of the liver, the right lung, the right arm, and the right side of the head and neck (Fig. 1). The right duct usually empties into the junction of the right internal jugular and subclavian veins.

## Physiology

In 1653 Rudbeck showed the anatomical relationships between the liver and intestinal lymphatics in the dog and correlated this with observations on parietal, peripheral and other visceral lymphatic trunks. Until recently, technical

TABLE II

THE PROTEIN AND FAT CONTENT OF CHYLOUS LYMPH: 41 PROTEIN DETERMINATIONS AND 56 FAT DETERMINATIONS FROM 63 REPORTED CASES

| Ref. | No. | Type | Protein | Fat | Ref. | No. | Type | Protein | Fat |
|---|---|---|---|---|---|---|---|---|---|
| 1. | 53 | CA | 3.9 | 1.3 | 32. | 86 | CT | 4.3 | 1.8 |
| 2. | 55 | CA | 4.4 | 1.0 | 33. | 94 | CT | — | 4.8 |
| 3. | 52 | CA | — | 9.2 | 34. | 122 | CT | 3.8 | — |
| 4. | 97 | CA | — | 3.3 | 35 | 133 | CT | 3.1 | 5.6 |
| 5. | 126 | CA | 2.2 | 4.7 | 36 | 133 | CT | 6.1 | 5.2 |
| 6. | 131 | CA | 4.1 | 4.1 | 37 | 138 | CT | 3.8 | — |
| 7. | 136 | CA | 4.3 | 4.0 | 38. | 158 | CT | 2.8 | .14 |
| 8. | 142 | CA | 1.2 | .65 | 39. | 166 | CT | — | 1.3 |
| 9. | 142 | CA | 1.3 | .33 | 40. | 168 | CT | 5.6 | 3.7 |
| 10. | 144 | CA | — | 3.9 | 41. | 191 | CT | — | .64 |
| 11. | 146 | CA | 1.2 | .01 | 42. | 191 | CT | — | 2.0 |
| 12. | 184 | CA | 1.0 | 1.3 | 43. | 191 | CT | — | 2.0 |
| 13. | 207 | CA | — | 1.4 | 44. | 191 | CT | — | 2.7 |
| 14. | 287 | CA | 4.5 | 3.6 | 45. | 191 | CT | — | 2+ |
| 15. | 290 | CA | — | 9.5 | 46. | 191 | CT | — | 1.2 |
| 16. | CH | CA | — | 2.0 | 47. | 191 | CT | — | .7 |
| 17. | 46 | CO | — | 1.0 | 48. | 223 | CT | — | 1.5 |
| 18. | 104 | CO | 3.5 | .80 | 49. | 236 | CT | 4.8 | — |
| 19. | 155 | CO | 4.5 | 2.0 | 50. | 236 | CT | — | .5 |
| 20. | 155 | CO | 3.7 | 2.3 | 51 | 234 | CT | — | 2.3 |
| 21. | 159 | CO | — | 4.0 | 52. | 253 | CT | 3.8 | 1.9 |
| 22. | 165 | CO | 4.5 | — | 53. | 270 | CT | 1.2 | 5.0 |
| 23. | 193 | CO | — | 1.9 | 54. | 277 | CT | 3.3 | 1.8 |
| 24. | 207 | CO | 11. | 11. | 55. | 284 | CT | 6.6 | — |
| 25. | 259 | CO | 3.9 | .56 | 56. | 291 | CT | 2.0 | 2.0 |
| 26. | CH | CO | 2.2 | 6.2 | 57. | 124 | CT | 2.3 | 2.8 |
| 27. | CH | CT | 2.2 | — | 58 | 124 | CT | 2.5 | 1.2 |
| 28. | 18 | CT | 5.8 | — | 59. | 124 | CT | 1.0 | 1.2 |
| 29. | 44 | CT | — | .83 | 60. | 124 | CT | 3.7 | .44 |
| 30. | 67 | CT | 2.7 | 1.8 | 61. | 124 | CT | 2.6 | 1.3 |
| 31. | 76 | CT | 6.1 | 25. | 62. | 124 | CT | 3.4 | 3.2 |

Average Values: Protein 3.38; Fat 3.04.

CA—Chylous ascites
CO—Chylous ascites and chylothorax combined
CT—Chylothorax
CH—Charity Hospital cases

hazards shunted further investigations away from the liver and intestinal lymphatics; accordingly, many of Rudbeck's observations have faded into the limbo of half-forgotten facts[155].

In man, the lymphatic circulation is rudimentary and many factors aid in the transport of lymph through the valved lymphatics. These factors include arterial pulsations, muscular activity, intestinal peristalsis and changes in intra-abdominal and intrathoracic pressure[271]. Ninety-five per cent of the volume of thoracic duct lymph comes from the liver and intestinal lymphatics to be blended with parietal and peripheral lymph in the *cisterna chyli* prior to evacuation through the thoracic duct[187]. All figures on human thoracic duct lymph flow are necessarily collected from individuals with lacerated or diseased thoracic ducts. These are estimates rather than normal values. Crandall and his co-

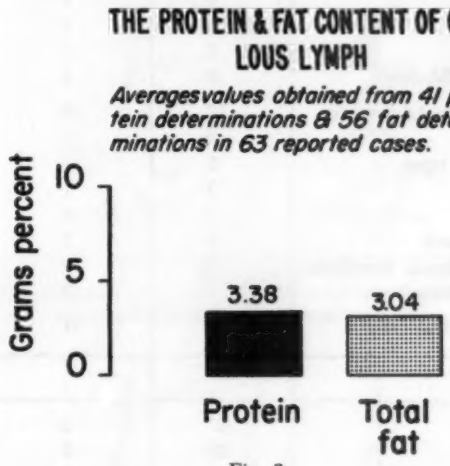


Fig. 2

workers estimated the rate of flow to be 0.93 c.c. per minute[53]. In the anesthetized dog the volume of lymph from the liver lymphatics, intestinal lymphatics and thoracic duct was equivalent in 24 hours to 47, 39 and 95 per cent of the estimated plasma volume respectively[189]. Starling's[246] contention that an increased flow of liver lymph follows obstruction of the inferior *vena cava* above the diaphragm was confirmed by Nix et al. In dogs with experimentally produced hepatovenous congestion and with cirrhotic livers, the flow of hepatic lymph was two to five times that found in normal dogs. The flow of intestinal lymph was not increased in these animals[189].

The chief function of the lymphatics and lymph is removal of excess blood protein from the tissues, maintenance of fluid balance, extrahepatic origin of globulin, and the absorption and transport of metabolic and ingested fat. As

TABLE III

ETIOLOGY OF CHYLOTHORAX AND CHYLOUS ASCITES
DATA OBTAINED FROM 302 SELECTED CASES

| Spontaneous | Chylothorax | Chylous Ascites | Combined |
|---|---|---|---|
| Extrinsic Lymphatic Obstruction | 24 | 64 | 15 |
| Malignant neoplasms | 15 | 16 | 8 |
| Benign neoplasms | 2 | 4 | 0 |
| Benign lymphadenophy | 5 | 15 | 4 |
| Peritoneal adhesions | 0 | 8 | 2 |
| Liver diseases | 2 | 15 | 1 |
| Volvulus | 0 | 1 | 0 |
| Intestinal obstruction | 0 | 1 | 0 |
| Hernia | 0 | 1 | 0 |
| Aneurism | 0 | 1 | 0 |
| Splenomegaly | 0 | 1 | 0 |
| Hydrops of gallbladder | 0 | 1 | 0 |
| Intrinsic Lymphatic Obstruction | 12 | 36 | 14 |
| Parasites | 0 | 1 | 0 |
| Neoplasms, all types | 5 | 23 | 11 |
| Stones | 0 | 1 | 0 |
| Tuberculosis | 0 | 6 | 1 |
| Congenital stenosis | 0 | 2 | 0 |
| Jugular or subclavian thrombosis | 5 | 0 | 2 |
| Thoracic duct thrombosis | 2 | 3 | 0 |
| Type Obstruction Unknown | 19 | 31 | 0 |
| Traumatic | | | |
| Penetrating | 37 | 2 | 0 |
| Surgical | 26 | 2 | 0 |
| Cervical | 2 | 0 | 0 |
| Thoracic | 24 | 0 | 0 |
| Abdominal | 0 | 2 | 0 |
| Nonsurgical | 11 | 0 | 0 |
| Stab wounds | 3 | 0 | 0 |
| Gunshot wounds | 5 | 0 | 0 |
| Explosive fragments | 3 | 0 | 0 |
| Nonpenetrating | 31 | 13 | 4 |
| Hyperextension of spine | 2 | 0 | 0 |
| Injury with fracture | 6 | 1 | 1 |
| Injury without fracture | 21 | 9 | 3 |
| Ruptured chylous cyst | 0 | 2 | 0 |
| Severe cough | 2 | 1 | 0 |
| Totals | 123 | 146 | 33 |

the liver and intestines contribute up to 95 per cent of the thoracic duct lymph[187], and as the primary function of lymph is the removal of excess blood protein from the tissues[69,246] the protein pattern of liver and intestinal lymph merits special interest. In dogs the protein content of hepatic, intestinal and thoracic duct lymph was found to be 78, 49 and 54 per cent of the protein concentration of plasma respectively[189]. In 1941 Brinkhous and Walker[30] found that thoracic duct lymph has lower values for fibrinogen and prothrombin than plasma and hepatic lymph. The diverse functions of lymph protein have long

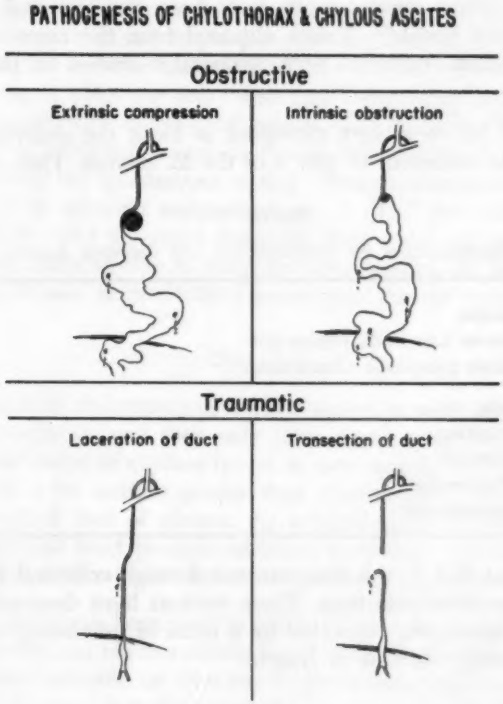


Fig. 3

been recognized. Antibodies and enzymes[13,26,96,118] have been reported in a concentration proportional to the protein content of the particular lymph sample[40,107,178,218]. The amylase activity of intestinal lymph is greater in fed than in fasting rats due partially to an increased lymph volume[23]. The intestines esterify large amounts of cholesterol and consequently, the cholesterol concentration of intestinal lymph is greater than plasma. Alkaline phosphatase is supplied to the plasma from the small intestine by way of the lymph[79]. Bile is necessary for the release and transport of phosphatase from the intestinal mu-

cosa[80]. In experimental intestinal obstruction, thoracic duct lymph flow remains normal[8], yet lecithinase can be detected in thoracic duct lymph[139].

The chemistry of chylous lymph in humans has received scant consideration in the literature. It is estimated by several authors that the specific gravity ranges from 1.012 to 1.020; total fat content, ½ gm. per cent to 3 gm. per cent; total protein content, 1 to 6 gm. per cent; and A/G ratio 3 to 1. Total fat content varies markedly with dietary fat. The total protein content remains relatively stable, approximately 3 gm. per cent (Table II, Fig. 2). Unfortunately, electrophoretic protein measurements have been demonstrated to be unsatisfactory for chylous lymph[151]. Values obtained from the cases reported in the literature are seldom controlled with comparable studies on the blood serum of plasma.

Blalock and his co-workers attempted to block the abdominal lymphatics in dogs and were successful in only 3 of the 52 animals. They attributed their

TABLE IV

CLASSIFICATION OF CHYLOTHORAX AND CHYLOUS ASCITES

| |
|---|
| I Spontaneous |
| A. Extrinsic Lymphatic Obstruction |
| B. Intrinsic Lymphatic Obstruction |
| II Traumatic |
| A. Penetrating |
| 1. Surgical |
| 2. Nonsurgical |
| B. Nonpenetrating |

failure to the fact that lymph was rerouted through collateral lymphatics and lymphaticovenous communications. These workers have demonstrated that the main lymphatic trunks are connected by a maze of interlacing collaterals, thus explaining the retrograde flow of lymph.

## CLASSIFICATION

Chylothorax and chylous ascites are classified as to mode of onset into spontaneous and traumatic (Tables III and IV). In spontaneous chylothorax, the etiology is predominantly obstructive and malignant. Lymphomas compress the thoracic duct by invasion through its wall and carcinoma involves the duct through its radicals.

Of the 123 cases of chylothorax, 55 (44.7 per cent) were spontaneous in origin. In these 55 cases, lymphatic obstruction was reported in 36 (65.4 per cent), was extrinsic in 24 cases (66.6 per cent), intrinsic in 12 cases (33.3 per

cent). The 68 cases (55.3 per cent) of traumatic chylothorax were divided into penetrating and nonpenetrating. Penetrating chylothorax was subdivided into surgical and nonsurgical. Twenty-six cases (38.2 per cent) of traumatic penetrating surgical chylothorax complicated 17 cases of cardiovascular surgery, 4 sympathectomies and 1 case each of esophageal, lung, posterior mediastinal, and neck dissection procedures (Table V).

One hundred forty-six cases of chylous ascites included 100 spontaneous (89.7 per cent) and 15 (10.3 per cent) traumatic. One hundred (76.3 per cent) of the spontaneous variety were due to obstruction 64, (64 per cent) of which were extrinsic obstruction and 36 (36 per cent) intrinsic thoracic duct or cisterna obstruction. The 15 cases of traumatic chylous ascites resulted from nonpenetrating trauma in 13 (86.6 per cent) instances, penetrating surgical trauma in 2 instances and penetrating nonsurgical trauma in no instance. Thirty-three (10.9 per cent) cases of chylothorax and chylous ascites combined included 29 (87.8 per cent) cases of the spontaneous variety. These spontaneous cases were all obstructive, due to extrinsic compression in 15 (51.7 per cent) and intrinsic compression in 14 (48.3 per cent) instances. Four (12.1 per cent) cases of the traumatic variety were all due to nonpenetrating trauma. No instances of penetrating surgical trauma or penetrating nonsurgical trauma were found.

## Diagnosis

The diagnosis of chylothorax or chylous ascites is rarely made before thoracentesis or paracentesis, and then only after much delay and confusion. The chemical indentification of chylous lymph is most specific. No other fluid in the human body has a fat content greater than plasma and a protein content approximately one-half that of plasma. As establishment of normal values and methods of diagnosis must precede advances in therapy, chemical data on reported cases of chylothorax and chylous ascites have been tabulated (Table II).

Lipophilic dyes have been used to label chylous lymph and make the diagnosis of chylothorax and chylous ascites by colorimetric methods[133,173]. Tasteless, nontoxic green and red cold tar dyes can be given with butter or oleomargarine. These dyes are absorbed into the lymphatic system as foreign substances where they are bound to the albumin fraction. Flat plate of the abdomen can be diagnostic of chylous ascites[290]. The liver is clearly delineated from the fluid as a smooth, radiopaque structure in comparison to the radiolucent fat containing ascitic fluid. In the absence of heavy metal ingestion and retention in the R.E. cells of the liver, this finding justifies the diagnosis of chylous ascites[290].

Radioactive drugs and lymphangiograms have been used in the study of lymphatic diseases of the extremities and might prove of diagnostic value at a later date[57]. $I^{131}$ albumin has been shown in animal studies to be a safe, stable compound absorbed largely via the lymphatics. Animals with lymphatic obstruc-

tion have shown flat absorption curves following the administration of this compound[57].

## Clinical Picture

The large volume of thoracic duct lymph flow explains the rapid development of chylothorax and chylous ascites and its recurrence after aspiration. The signs and symptoms are those of mechanical compression and plasmapheresis with loss of a large volume of chylous lymph. In the cases of chylothorax included in this report the effusion was right-sided in 53 (45.7 per cent) instances, left sided in 42 (36.2 per cent) instances, bilateral in 21 (18.1 per cent) instances and unknown in 7 instances. The most prominent symptom of chylothorax in this series was dyspnea; the most prominent symptom of chylous ascites was abdominal enlargement with abdominal pain; and the cases of combined chylothorax and chylous ascites showed both dyspnea and abdominal enlargement with abdominal pain.

TABLE V

Surgical Procedures Complicated by Chylothorax

| Surgical Procedure | Number of Cases |
|---|---|
| Cardiovascular surgery | 17 |
| Tetralogy of Fallot | 10 |
| Tricuspid atresia | 3 |
| Pulmonary stenosis | 2 |
| Vascular ring | 1 |
| Coarctation of aorta | 1 |
| Sympathectomy | 4 |
| Neck dissection | 2 |
| Esophageal resection | 1 |
| Posterior mediastinal dissection | 1 |
| Lysis of lung adhesions | 1 |
| Total | 26 |

## Treatment

*Chylothorax:*—In the past, treatment of chylothorax has been largely symptomatic with repeated thoracentesis and chylous replacement orally or intravenously[199]. Efforts to compress the thoracic duct by pneumothorax[229], phrenic crush[67] and rib resection[74,154,235] and attempts to obliterate the pleural space by the injection of irritants[94,166,279,224] have been largely unsuccessful. By way of prevention, accidental partial or complete transection of the thoracic duct complicating surgery for other conditions is better handled by thoracic duct ligation

distal to the laceration, than by lymphangeoplasty or lymphaticovenous anastomosis. Lampson in 1948 reported the successful treatment of chylothorax by thoracic duct ligation through a thoracotomy approach. As the thoracic duct has been removed surgically without undesirable sequelae[167], there is little reason to fear the development of chylous ascites as a postoperative complication of thoracic duct ligation for traumatic chylothorax. Conservative treatment[72], employing repeated aspiration or continuous thoracotomy drainage, may be tried as long as the daily volume of aspirate decreases. Thoracic duct ligation, however, should never be unduly delayed. The use of lymphaticovenous shunts from the thoracic duct to the azygos vein, with or without internal prosthesis, has been reported in animals[62]. The probability of intraluminal obstruction of the anastomosis makes this difficult procedure less desirable than simple effective thoracic duct ligation. In the 123 cases of chylothorax in this report, 68 (55.3 per cent) were of traumatic origin; 19 (28 per cent) of these traumatic chylothorax cases were treated by thoracic duct ligation with 2 deaths.

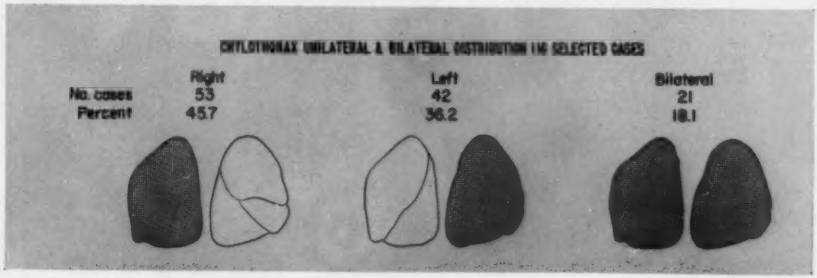


Fig. 4

*Chylous Ascites:*—One hundred thirty-one (89.7 per cent) of the 146 cases of chylous ascites included in this report were of spontaneous or obstructive origin. Twenty-nine (87.8 per cent) cases of the 33 combined chylothorax and chylous ascites cases were of spontaneous or obstructive origin. As malignancy is the most common cause of thoracic duct or cisterna obstruction, the treatment of chylous ascites is the treatment of the effect rather than the cause. Conservative therapy consists of repeated paracentesis with oral or intravenous replacement of chyle. Auxiliary lymphatic drainage operations have been tried, employing a variety of saphenous peritoneal anastomoses[16,94] and drainage of the peritoneal cavity into subcutaneous tissues of the thigh or abdomen through the use of drains or buttons[94,117]. These operations offer palliation at best. As chylous ascites can be produced by a benign neoplasm obstructing the *cisterna chyli* and as the general prognosis is poor, exploration of the region where the superior mesentery artery crosses between the pancreas and the third portion of the duodenum, has been advocated for cases of doubtful etiology[53]. Possibly the use of an open loop of transected ileum attached to the right upper abdominal wall with the mucosal surface intraperitoneal might be considered. It has

been shown that an isolated loop of intestine will absorb injected nonchylous ascitic fluid. It remains to be shown that chylous lymph can be absorbed by this intestinal transplant and transmitted to the systemic venous system or right thoracic duct, when the *cisterna chyli* and thoracic duct are obstructed. No proven definitive method of treatment for chylous ascites gives as satisfactory a result as thoracic duct ligation for chylothorax.

## Mortality

Of the 123 cases of chylothorax, 55 (44.7 per cent) were reported as cured. Of the 146 cases of chylous ascites, 52 (36.3 per cent) were reported as cured. Of the 36 cases of chylothorax and chylous ascites combined, 4 (11.1 per cent) were reported as cured. Nineteen cases of traumatic chylothorax were treated by ligation of the thoracic duct and 17 (89.4 per cent) of these were reported as cured.

## Summary

1. Chylothorax and chylous ascites are but sequelae of an internal lymph fistula.

2. The incidence of these conditions is rising with the increase in such procedures as aortic grafts, cordiovascular surgery, and hepatic and pancreatic resections.

3. The thoracic duct is variable throughout its course with the exception of the segment between the levels of T 8 and T 12.

4. Lymphatic physiology is a complex field and has not yet received adequate study.

5. Chylothorax and chylous ascites are classified as either spontaneous or traumatic.

6. The diagnosis is established by the chemical indentification of chylous lymph.

7. The clinical picture results from a combination of mechanical compression and chylous fluid loss.

8. Chylothorax is best treated by ligation of the thoracic duct; a definitive treatment for chylous ascites remains to be developed.

The lymphatic network is a primitive venous system and diseases of the lymphatic trunks closely resemble diseases of the veins themselves. As the thoracic duct is the orphan of vascular surgery, the surgical therapy of lymphatic disease can best be envisioned by comparison with similar venous entities. With femoral or saphenous vein obstruction, increased intravenous pressure, incompetency of the valves, and dilatation and distortion of the distal veins

result. Likewise with thoracic duct obstruction, increased intralymphatic pressure, valvular incompetency, lymphangiectasis, and retrograde circulation of lymph ensue. Internal lymph fistulae with resultant chylous effusions follow:

1. Trauma to the lymphatic trunk with either transection of the trunk or laceration without loss of continuity of the trunk.

2. Obstruction of the thoracic duct, cisterna chyli and collateral lymphatics with a resultant increased intralymphatic pressure and pressure necrosis of the lymphatic wall.

Treatment of the resultant chylothorax or chylous ascites is dependent on the cause. As the lymphatics themselves are normal in traumatic lymph fistulae, the prognosis is better and the treatment is more clearly defined.

## Discussion

*Dr. O. H. Wangensteen:*—It is a pleasure to have the opportunity to discuss this nice and informative paper. This subject has keen interest for most surgeons.

At the turn of the century, one heard a great deal about surgical injuries of the thoracic duct, especially after operations in the neck, such as block dissection for cancer. I recall that Harvey Cushing mobilized one of the jugular veins and implanted the injured thoracic duct into the vein. Some years later, Percy Sergeant of London, found that following ligature of the thoracic duct in the upper thorax of the dog, chyle still would find its way into the venous system.

Packing of the wound in the neck for a chylous fistula became general practice, because it was the simplest procedure and often quite effective. Today, as Dr. Nix said, Blalock's operation for mobilization of the subclavian artery in the shunt operation for Tetralogy of Fallot is one of the commoner causes of fistula of the thoracic duct. Maloney, a previous associate of Dr. Blalock's stressed the circumstance that a thoracic duct fistula will respond usually to conservative management. [Maloney, J. V., Jr. and Spencer, F. C.: Nonoperative Treatment of Traumatic Chylothorax, *Surgery 40*:121-128 (July), 1956.] I would infer from reported data, however, that operative management is followed by a quicker convalescence. I think in those instances of chylothorax where the response to conservative treatment is slow, following employment of either a needle or a catheter, surgeons should consider ligating the thoracic duct above the diaphragm. It is encouraging to know that such a procedure can be done safely and without harm to the patient—an extension of the findings of Percy Sargeant concerning the safety of ligature of the thoracic duct in the upper thorax.

Dr. Nix undoubtedly applied a good deal of study to this problem to give us this very nice presentation. He is therefore in a much better position than I

to discuss this with you in an intelligible manner. There is, I know, a tendency at the moment in the majority of such cases following vascular injury, (and in our clinic too), to treat chylothorax conservatively. Moreover, as I recall it, in the Maloney series from the Blalock Clinic there were no deaths.

It is always a mystery to me that one can grub out an adherent esophageal cancer in the upper thorax without provoking a persistent thoracic duct fistula. Perhaps I have failed to recognize its presence. It would appear that it should happen now and then.

*Dr. I. Snapper:*—Dr. Nix's investigation is of great importance for many fields of medicine, because the study of the connections of the lymphatics of the abdomen is of great clinical interest. This becomes evident in the pathogenesis and treatment of pyelitis. The colon bacilli travel from the intestine via the lymphatics to the kidney.

Forty years ago when there were of course no antibiotics, Dr. Bumm, the obstetrician from Berlin, remembered that colon bacilli grow profusely upon milk. At that time pyelitis of pregnancy was always treated with a milk diet. Bumm then put the pregnant patients with pyelitis on a milk-free diet and saw to his great satisfaction that on this revolutionary diet many such patients did very well.

It may be that at least part of the success of the modern treatment of pyelitis with antibiotics is due to the sterilization of the intestine which prevents bacterial infection of the kidneys via the connecting lymphatics.

In olden times distinctions were made between chylous fluid and chyliform fluid. Chylous fluid was due to the presence of chyle, chyliform fluid was a fine emulsion of protein particles.

In later years this distinction was no longer made. I wonder whether Dr. Nix has any information about chyliform fluid.

*Dr. Wangensteen:*—Dr. Snapper, I should like to ask why milk is dangerous.

*Dr. Snapper:*—You can't grow colon bacilli better than on milk. Thus during a milk diet the colon bacillus population of the colon is especially heavy which increases the chances of infection of the renal pelvis.

*Dr. James T. Nix:*—Let me first answer the question raised by Dr. Wangensteen. Thoracic duct lymph can be labeled by giving a high fat diet the evening before operation. Recently, Klempner advocated the preoperative use of tasteless coal-tar dyes that make thoracic duct lymph either red or green at the time of operation; and in liver surgery, the red-coal dye could be used to distinguish postoperative extravasated lymph from bile.

Dr. Snapper's question is well taken, and this was the reason for the reference in this paper to nomenclature of the lymph system, as there is much con-

fusion in the literature in the use of "chyliform", "chylous" and "pseudochylous". All the cases included in this report showed one or more of the following criteria: 1. free fat microscopically, 2. chemical identification of chylous lymph by a high fat and low protein content, and 3. autopsy or surgical evidence of lymphatic fistulae.

Chyliform fluid is normally a fluid of degeneration, containing microscopic particles of protein and neoplastic tissue. This fluid shows a high protein and a low fat content. The fat content will probably be lower than that of plasma and free fat will not be demonstrable microscopically.

*(Editor's Note: Limitations of space make it necessary to omit the references. They will, however, appear in the reprints.)*

# MANAGEMENT OF GASTROINTESTINAL BLEEDING*

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Philadelphia, Pa.

Gastrointestinal bleeding frequently presents a complex problem both for diagnosis and treatment. In a broad sense this bleeding can occur as hematemesis and/or melena and vary in degree from minimal streaking of blood to rapidly fatal exsanguinating hemorrhages. The acute variety occurs more frequently and has attracted more attention but the chronic form at times can represent an equally difficult challenge for management. The most common causes producing gross to massive bleeding are ulcer and esophageal varices. Less frequent causes are gastritis, neoplasm of the stomach, hiatal hernia, blood

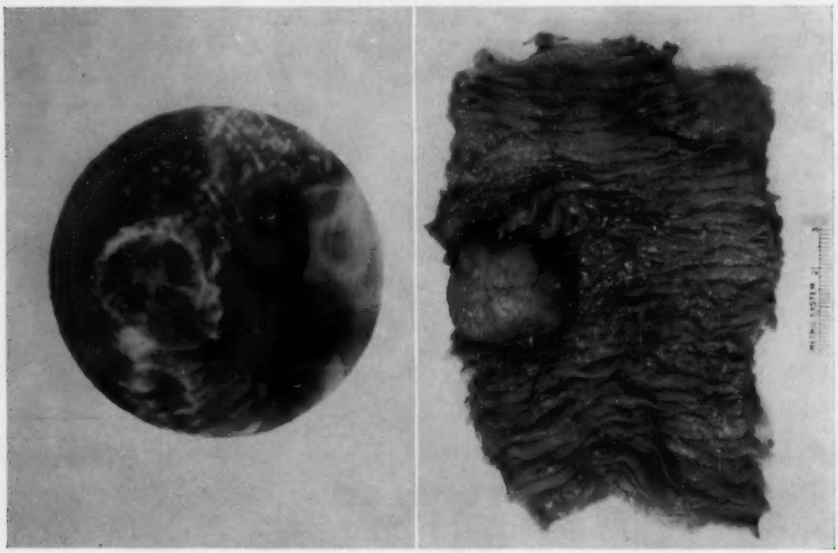

Fig. 1a



Fig. 1b

dyscrasias, neoplasm (Fig. 1), and inflammations of the small (Figs. 2a, b) and large intestine (Fig. 3) and pancreas, and hereditary hemorrhagic telangiectasia. In addition, there are conditions which produce mild to severe bleeding such as eosinophilic gastritis, bleeding due to certain drugs such as cortisone (Fig. 4), aspirin and Butazoladin, prolapse of the gastric mucosa, acute febrile

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diseases (yellow fever, acute yellow atrophy of the liver, severe Weil's disease), intracranial lesions resulting from trauma, hemorrhage, abscess or tumor, gastric arteriosclerosis, Mallory-Weiss syndrome, Curling's ulcer, Henoch's purpura, and mesenteric vascular occlusion.

One would assume from the numerous reports in the literature that the management of these cases was uniformly agreed upon. Unfortunately there is still sufficient divergence of opinion in some quarters to lead to confusion in management. There are proponents of exclusive medical management and others who feel that an excessive mortality can only be lowered by prompt surgical intervention. Meulengracht[1] has reported a mortality of only 2.5 per cent in a large series of bleeding ulcer patients treated by an early feeding regimen and supportive measures. Miller[2] and his colleagues feel that an early feeding regimen is superior to surgical intervention. On the other hand Finsterer[3] and Gordon-Taylor[4] have long advocated surgery to reduce a high mortality rate and

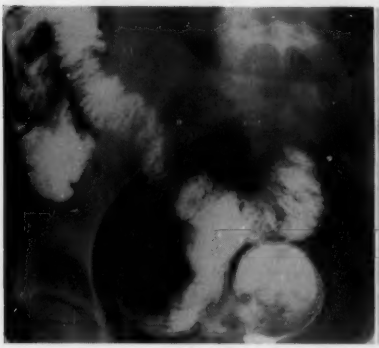

Fig. 2a

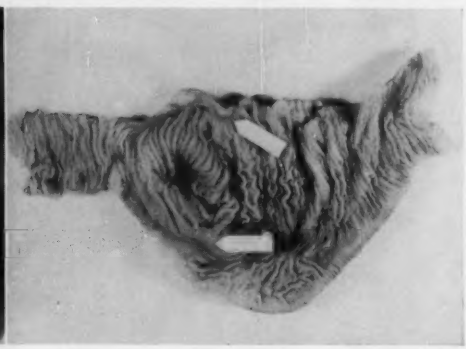

Fig. 2b

Stewart[5] in this country recommends that all patients with massive bleeding from ulcer should have immediate surgery.

A third method of handling these patients is a combined medical-surgical approach at times called team management. From our experience we have been impressed with the importance of having a systematic plan for handling these patients and prefer to employ team management (Fig. 5). Accordingly all patients with gross gastrointestinal bleeding coming to our receiving ward are transferred to the medical service where they are seen promptly by a resident especially assigned to the bleeding team. After obtaining a history and performing a physical examination consistent with the patient's condition, he notifies members of the team or their designated representatives at any time of the day or night. Each patient is treated medically unless he continues to bleed or rebleeds after a period of cessation, in which event he is considered a candidate for prompt surgical intervention (Fig. 6).

## Diagnosis

It is generally recognized that massive bleeding in the elderly patient is a major emergency requiring prompt treatment. Ordinarily less attention is given to the patient with mild to moderate bleeding, but careful supervision of the latter is equally important because of the frequency with which recurrent bleeding may throw him into shock a few hours after admission. Experience has shown that the majority of patients are admitted during the early or late even-

TABLE I

Diagnosis—Treatment—Mortality
285 Patients with Gross Gastrointestinal Bleeding

| | Total | Medical Treatment | Medical Mortality | Surgical Treatment | Surgical Mortality |
|---|---|---|---|---|---|
| Duodenal ulcer | 112 | 68 | 10 | 44 { E-16 / I-28 } | 8 |
| Gastric ulcer | 28 | 16 | 6 | 12 { E-2 / I-10 } | 1 |
| Marginal ulcer | 11 | 8 | 1 | 3 { E-1 / I-2 } | 0 |
| Esophageal varices | 43 | 28 | 14 | 15 { E-5 / I-10 } | 4 |
| Unknown site | 32 | 29 | 4 | 3 { E-2 / I-1 } | 0 |
| Others* | 30 | 20 | 7 | 10 | 0 |
| Neoplasm stomach | 14 | 1 | 0 | 13 | 1 |
| Hiatal hernia | 9 | 6 | 0 | 3 | 0 |
| Blood dyscrasias | 6 | 3 | 0 | 3 | 1 |
| Totals | 285 | 179 | 42 | 106 | 15 |

*Neoplasm or inflammation of small and large intestine and pancreas; hereditary telangiectasia.
E—Emergency
I—Interval

ing when a reduction in staff and in routine facilities adds to the confusion of management. It is for this reason, as much as any other, that a systematic plan of management should be set up with which all hospital personnel are familiar.

It is essential to know the severity and site of bleeding as early as possible; consequently the first step in evaluating a patient admitted for gastrointestinal bleeding is to determine if he is in shock from blood loss. For this one may

depend upon the blood pressure and pulse considering a systolic blood pressure of 90 mm. of mercury or below and/or a pulse of 110 beats per minute or above as indicative of incipient shock and of the need for blood replacement. It should also be borne in mind that there is an occasional patient, who even though he is suffering from great blood loss, fails to manifest tachycardia or a fall in the systolic pressure but will show gradual widening of the pulse pressure. The general condition of the patient offers additional valuable diagnostic information; pallor, a cold, moist skin, air hunger, thirst and restlessness are important secondary signs of bleeding. The hematocrit, although routinely performed, is

Fig. 3

rarely an index of the blood loss because of hemoconcentration. Blood volume determination using Evan's blue dye, does not appear to be a practical aid in determining the patient's need for blood under these emergency conditions. Recent experience with radioactive labeled albumin, however, suggests that it may soon be put upon a practical basis for this purpose.

The next important step is one of exact diagnosis. Although peptic ulcer is the commonest cause of gastrointestinal bleeding, Table I shows that only a little over one-half of the 285 patients seen by our bleeding team had an ulcer as the cause of their bleeding. It is possible that the number of those listed as

"site unknown" bled from superficial ulceration or erosion, but this was only established in three coming to surgery (Fig. 7). It is obvious that there are many causes for serious bleeding other than ulcer and therefore definite diagnosis is an important preliminary to treatment. The majority of the patients coming to the hospital with bleeding are unable to give a history which permits definite diagnosis. A thorough physical examination is therefore performed, a special search is made for the presence of icterus, purpura, angiomata of the skin, hepatosplenomegaly and lymphadenopathy, and the condition of the gums and the mucous membranes of the mouth is carefully observed. An inspection of the perineum and a digital rectal examination should be performed, with benzidine testing of the stool to confirm the presence of melena. If the history of hematemesis is not well established, a Levin tube should be passed to aspirate

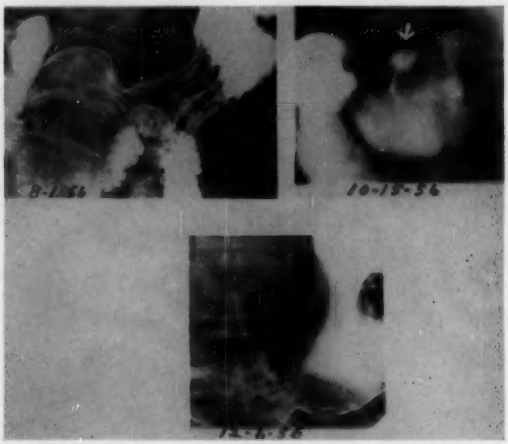

Fig. 4

the gastric contents. For all practical purposes the presence of gross blood in the stomach establishes the bleeding site somewhere proximal to the ligament of Treitz. This is of particular diagnostic importance when roentgen studies fail to show a lesion. When stigmata are present suggesting portal hypertension with esophageal varices as the site of bleeding, a tube of the Tocantins or Senstaken type fitted with a balloon for tamponade should be used. This permits compression of the varix, aspiration of the stomach and the introduction of liquid feedings if desired. If blood can still be aspirated after one to two hours' tamponade, the bleeding point should be suspected to be below the esophageal varix, although occasional inadequate tamponade may produce this. It should be recalled that the incidence of peptic ulcer in the cirrhotic patient is approximately three times that in the noncirrhotic and that ulcer can often be the site of bleeding even when large varices are present in the esophagus. Under these circumstances the use of a tube with balloon tamponade may be

of great assistance in arriving at a correct diagnosis. An unusual case of gastric varix is shown in Figure 8.

A complete blood count, urinalysis, blood typing and crossmatching and bromsulfalein excretion test of liver function are the emergency laboratory procedures which should be available at all times. The latter test has been of considerable aid to us in evaluating the patient in whom cirrhosis of the liver is suspected. We give 2 mg. of the dye per kilo of body weight and normally find all dye will be removed from the blood within 30 minutes, but when hemorrhage is superimposed on underlying hepatic disease a varying degree of retention will be found in the majority of patients. The electrocardiogram and serum amylase determination will be required at times on an emergency basis

Fig. 5

Fig. 6

but the blood urea nitrogen, carbon dioxide combining power and electrolyte studies are performed the following day when indicated.

All patients in whom the bleeding is suspected to be arising proximal to the ligament of Treitz should have a prompt upper gastrointestinal roentgen study as soon as their condition permits. The majority will be stabilized and free of shock within 12 to 15 hours and can be studied the morning after admission. Selective positioning of the patient and air contrast are used avoiding undue pressure or manipulation of the abdomen.

Esophagoscopy and gastroscopy will often prove helpful and may be employed in selected cases when roentgen study fails to show a lesion.

## Case Reports

*Case 1:*—M. L. S., a 24-year old white female who had had weakness and anemia for two years requiring four to six transfusions yearly was found to have stools that were constantly positive for occult blood, and on roentgen study a defect in the mid-jejunum suggestive of a small bowel tumor (Fig. 1a). A walnut-sized leiomyoma that was the site of bleeding from the small bowel was found (Fig. 1b). This patient made a satisfactory convalescence and has had no further evidence of bleeding or anemia.

*Case 2:*—B. T., a 23-year old white female had complained of severe weakness and anemia requiring three to four transfusions of whole blood yearly for three years prior to admission to the Jefferson Hospital. Although gastrointestinal roentgen studies had been performed previously, they had not revealed

Fig. 7

the site of bleeding. Our roentgenologist had the good fortune to find the unusual lesion seen in Fig. 2a. He was unable to be certain of its character but suggested the possibility of a Meckel's diverticulum or a duplication of the small bowel. Figure 2b shows the resected specimen of small bowel to be a simple large dilatation involving a segment of some 15 cm. and when it was opened there was a deep penetrating ulcer from which the bleeding was arising.

*Case 3:*—J. C., a 62-year old white female who had massive melena 36 hours before admission. She claims to have had no preceding symptoms of indigestion or abnormal bowel function. Roentgen study revealed a segmental defect in the ascending colon which was suspected of being neoplasm or segmental ulcerative colitis (Fig. 3). Proctoscopic examination was negative. Surgical intervention was contemplated but since we had had a similar case one week prior involving the mid-transverse colon which upon resection showed only benign

inflammation, it was decided to temporize. This patient became asymptomatic on medical treatment and a repeat barium enema showed a return to a normal appearance. She has been followed for three years since this episode and has been entirely free of symptoms.

*Case 4:*—M. G., 64-year old white female who was admitted to the hospital with a history of right upper quadrant pain referred to the subscapular area. The liver function test profile suggested obstructive jaundice but at laparotomy no evidence of extrahepatic obstruction was found. Liver biopsy showed the presence of cholangiolytic type cirrhosis. Postoperatively the patient had a stormy course with the serum bilirubin increasing up to 24 mg. per cent. There

Fig. 8

was severe anorexia and weight loss and the patient had to be fed entirely parenterally. Because her condition was desperate, Meticorten 30 mg. daily was administered with some improvement. This was gradually increased over a period of six weeks until she was getting 80 mg. per day. At this point she developed melena and a repeat upper gastrointestinal x-ray showed a large gastric ulcer in the antrum. In Figure 4 it will be noted that the stomach appeared normal on roentgen study preoperatively and before steroid therapy. After withdrawal of the steroid therapy and medical treatment for the ulcer, it gradually disappeared; the repeat roentgen study approximately two months later again showed a normal stomach.

*Case 5:*—G. Y., a 48-year old robust laboring man was admitted to the hospital because of the sudden onset of massive gastrointestinal bleeding 24 hours before. At the time of admission, he was in mild shock and responded to rest, sedation, and three units of whole blood. Gastrointestinal x-ray studies did not reveal the site of bleeding and later the patient rebled. A recommendation of surgical intervention was made but the attending staff on the medical ward did not agree that this should be done at this time. The patient received additional blood but subsequent rebleeding bouts followed and the patient ultimately demised. At necropsy a leiomyoma was found which seemed to exist almost entirely outside the lumen of the bowel with only a small portion projecting into the mucosal surface (Fig. 5). It was from this point that the bleeding was occurring. In retrospect it was felt that surgical intervention was indicated at

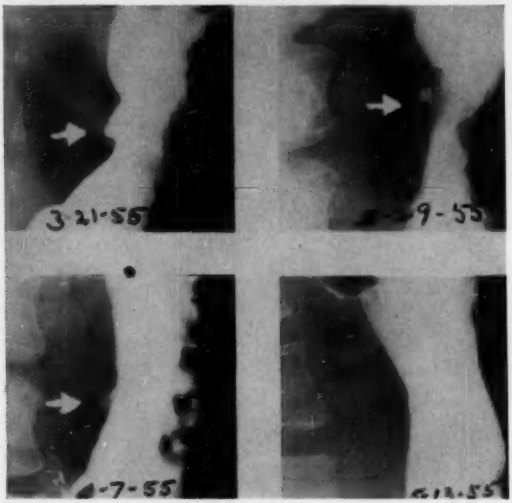


Fig. 9

the time of the first episode of rebleeding in the hospital and that this lesion would have been amenable to resection.

*Case 6:*—R. A., a 43-year old white male who had subtotal gastric resection five years previously for bleeding duodenal ulcer. Bleeding was massive upon admission and consisted of both hematemesis and melena. In spite of the transfusion of four units of whole blood within 24 hours, the patient did not appear to cease bleeding and a prompt subtotal gastric resection was performed. His convalescence was uneventful. Figure 6 shows the two-minute marginal ulcerations from which this massive bleeding was occurring.

*Case 7:*—F. E., a 53-year old white female who had protracted bleeding which was mostly melena. It was felt that a pyloric ulcer was shown on roentgen

study but at laparotomy no lesion was demonstrated (Fig. 7). In retrospect it is suspected that this finding represents the so-called "pyloric star".

*Case 8:*—A. R., a 51-year old white female who had moderately severe hematemesis and melena. The liver was palpable 4 cm. below the right costal margin on deep inspiration. Bromsulfalein test showed 15 per cent dye retained. The roentgen study suggested a neoplasm probably benign on the high lesser curvature (Fig. 8). Gastroscopic examination showed a tumor in this area but the surface appeared to be that of a normal gastric mucosa. At operation this lesion proved to be a large gastric varix which was satisfactorily ligated.

*Case 9:*—L. M., a 49-year old white female who had considerable blood loss due to hematemesis and melena but was admitted to the hospital without shock and was found to have a large mid-lesser curvature gastric ulcer. Treatment by continuous drip of Sustagen alternating with Maalox appeared to facilitate hemostasis. Bed rest and three units of whole blood were given. Satisfactory healing of the ulcer is shown in Fig. 9. The patient has been followed for two years since discharge and has had no recurrence of symptoms.

## Treatment

All patients should be given an initial trial on medical management which includes whole blood transfusions when indicated, bed rest, sedation and early feeding. Since most of these patients are free of pain but are very apprehensive, it is preferable to give sodium phenobarbital, grains 2 intramuscularly every six hours in the average adult and avoid the use of narcotics. Alkalis and anticholinergics are not often required but may be employed. When using the latter the potential side-effects on the cardiovascular and urinary systems should be looked for, particularly in the older age group. Oxygen by nasal catheter or tent will be required in some patients. Dehydration and urinary output may be used as a guide for fluid and electrolyte administration. As soon as the patient is free of nausea and asks for food he may be given one ounce of milk hourly with a gradual increase in the volume until soft foods are added promptly according to the tolerance of the patient.

More recently, based upon our investigative findings dealing with the effectiveness of complete buffering in checking bleeding from the stomach, we have introduced a Levin tube as early as possible into the stomach for gastric aspiration and subsequent feedings which were designed to produce as complete a buffering of the gastric juice as possible. After testing a variety of agents, we preferred a protein dextri-maltose mix known as Sustagen* because it could be given in sufficiently large amounts to produce adequate buffering and yet it was well tolerated by the majority of patients (Fig. 9). It had the advantage of being a nutriment of high caloric value with vitamin and mineral supple-

*Mead Johnson and Co., Evansville, Ind.

ments and yet it rarely produced abdominal cramps or diarrhea as had some protolysate products in our experience. To obtain adequate buffering we administered 150 to 200 c.c. of a 1:2 dilution of this substance every one to two hours around the clock. The ability to maintain buffering throughout the 24 hours appealed to us as an excellent means of combatting the deleterious effect of high acid and pepsin on hemostasis. In some patients, however, in order to overcome the extremely high acid peaks, an aluminum hydroxide, magnesium hydroxide gel (Maalox†) could also be given in 15 to 30 c.c. amounts between the Sustagen feedings. This regimen is usually employed when the patient is admitted at night and in the morning the gastric contents are aspirated through the Levin tube and the latter removed from the stomach just prior to roentgen examination. If ulcer is confirmed by x-ray, a small plastic nasogastric tube is then reinserted for continued tube feedings.

Following maximum improvement elective surgery should be recommended, based essentially upon the age of the patient and the frequency of complication of his ulcer. Operation was rarely recommended in a patient under 40 years of age in whom there was not a history of repeated complication. Those individuals in whom recurrent bleeding has occurred, but in whom the site cannot be found in spite of careful survey, may require so-called "blind" resection of the stomach. These patients are usually in the second or third decade, have few if any symptoms of indigestion, a high gastric acidity and a long history of recurrent hematemesis and/or melena. When properly selected, this type of bleeding patient usually responds well to surgical treatment.

When bleeding from ulcer continues or recurs in spite of supportive measures, the patient should immediately be considered a potential candidate for prompt surgical intervention. This decision forms the crux of team management because we have observed, as have others, that mortality rises very sharply in those patients who continue to bleed or rebleed after admission to the hospital, particularly when they are 45 years of age or over. Consequently these patients are kept under constant observation during their hospital stay and if rebleeding occurs, prompt re-evaluation must follow.

Tube feedings of a protein dextri-maltose mix also can be given to those patients with esophageal varices as long as balloon tamponade is maintained. Usually these patients can be brought into a state of stabilization within 24 to 72 hours and at this time they can be prepared for elective transesophageal varices ligation. When recovery from this operation is adequate the patient then can be evaluated for portacaval shunt.

Bleeding due to gastric neoplasm, hiatal hernia, Mallory-Weiss syndrome, Curling's ulcer and mesenteric vascular occlusion will regularly require surgical treatment, while patients with eosinophilic gastritis, hereditary telangiectasia

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and prolapse of the gastric mucosa should be operated upon only after all other measures of control fail. In most instances bleeding due to drugs, infections, blood dyscrasias and intracranial lesions can be handled medically.

Patients with inflammatory or neoplastic lesions of the small or large bowel usually stop bleeding after receiving general supportive measures. When their condition improves sufficiently, a careful roentgen study may be performed and in some instances Miller-Abbott tube passed to determine the site of bleeding. Medical or surgical treatment is then undertaken depending upon the diagnosis.

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## Discussion

*Dr. I. Snapper:*—About 25 years ago Sir Arthur Hurst, the famous gastroenterologist from London, published his statistics which showed that only less than 3 per cent of his patients with upper gastrointestinal hemorrhage died. At that time the blood transfusion was just being introduced into the practice of medicine and only small amounts of blood, in most cases even only saline, were given. Thus any method which leads to a fatality in upper gastrointestinal hemorrhage exceeding 3 per cent should be looked at askance.

A patient with an ulcer hemorrhage should be kept in bed, should not be carried around too often to an x-ray apparatus, should either receive milk diluted with lime water, or occasionally a Meulengracht diet. The amount of blood given should be limited and should not exceed the quantity which is necessary to keep the patient out of shock.

When every patient with an upper intestinal hemorrhage *must* receive so much blood that the hemoglobin climbs to 10 gm. per cent or higher, and the systolic blood pressure to 120 mm. hemoglobin or higher, then the danger for continuous hemorrhage is markedly increased. The amounts of blood which are necessary to guarantee the magic figures for hemoglobin and blood pressure mentioned above, are so great that a considerable rise of circulating blood volume results. This hypervolemia causes hemorrhage as soon as a weak spot in the circulatory tree is present anywhere. This has been clearly demonstrated by the frequent hemorrhages from esophageal varicosities which occur after albumin injections in patients with liver cirrhosis; the albumin injections cause

an increase of the circulating blood volume and hemorrhage ensues, even in patients who have never had an esophageal hemorrhage previously.

In other words, if the fatality of upper gastrointestinal hemorrhage exceeds 3 per cent, iatrogenic or chirurgogenic influences are to be blamed.

*Dr. O. H. Wangensteen:*—I was so fascinated by what Dr. Wirts had to say, I forgot there were questions to be answered.

There is no Utopia in this problem. In our institution I have been hoping for years that we could have an earlier go at some of these bleeding cases—that is to see the patients on admission to hospital.

Dr. Dennis, an erstwhile surgical protégé of mine, tells me he has established a cooperative medicosurgical supervision program with Dr. Andresen in his new place of work at the Kings County Hospital in Brooklyn. Moreover, Dr. Dennis tells me that the accomplishment of the surgeon in operating early has been no better than that of the internists, who accept and manage every other case admitted. John Stewart of Buffalo recently published his results with an actual program of early surgical intervention [*Surg., Gynec. & Obst.* **103**:409-415, (Oct.), 1956]. The results were certainly no better than those recorded by more conservative observers. These reports are a bit discouraging to the surgeon who would like to be somewhat more energetic in the management of massive hemorrhage and I confess to count myself in that group—without having had the opportunity to do so.

In 1944, (*Canad. M. A. J.* **53**:309, 1945) when I proposed and carried out so-called empiric resection for massive hemorrhage in patients who had hematemesis, and no lesion was found at operation, the operation did not find ready acceptance with my colleagues in my own institution. I am, however, pleased to note that this suggestion in the last dozen years has found favor amongst surgeons and that empiric gastric resection for massive hemorrhage with hematemesis has been taken up in many areas of our country.

Actually this situation of hemorrhage without a palpable or detectable lesion, being found on inspecting the stomach from the outside, at autopsy, was well known to French and German pathologists 50 years ago. One of the many roles of the surgeon is to try to lengthen life and to make it happier. I think there is a definite role for surgery in such cases of massive hemorrhage with hematemesis today. Some surgeons, notably Drs. Maloney and Spencer of Johns Hopkins advise a 7-inch long gastrotomy incision in the stomach through which the stomach and duodenum can be inspected for erosions or other occult sources of bleeding.

Dr. Maloney (now of Los Angeles) and I had the opportunity of discussing this matter recently in a panel discussion at the Clinical Congress of Surgeons in San Francisco. I asked him whether he had resected the stomach when

he failed to find a lesion under these circumstances. My recollection is that he said he omitted performing an empiric or blind gastrectomy in such an instance because no bleeding point was seen and that the patient died (Surgery 40:904, 1956). Once I was disappointed in not seeing a small erosion at the upper end of the resected specimen upon performing empiric resection for massive hemorrhage with hematemesis, but was heartened to learn that the pathologist found a microscopic diffuse ulcerative gastritis. Bleeding did not recur in that patient.

Now, as to what I would do in massive gastrointestinal hemorrhage. I would not operate willy-nilly on every patient who came in bleeding. I think the surgeon has to get his bearings concerning his patient. If the patient was in or had been in shock, I would be led to believe that there was arterial bleeding. When a patient bleeds down to shock level and has an erosion—even a small erosion, the bleeding is usually arterial or arteriolar in nature. I have seen a tiny cherry-red column rise as high as 40 cm. from such a source when the source of bleeding could not be felt from outside the duodenum or stomach. That height is about the level of arteriolar blood pressure. Venous bleeding is not like that. A really active hemorrhage is *always* arterial in origin, and if shock continues and sweating is present, the patient is a candidate for operation. That about summarizes my attitude toward the problem of massive hemorrhage accompanied by hematemesis in ulcer. Of course, I want to be informed of the patient's physical status too.

Dr. Snapper and I apparently disagree over the utility of blood transfusion in ulcer hemorrhage. I prefer to have a patient with a reasonable blood volume. Moreover, I like to have the patient come to surgery before the lag phases of hemorrhage and waiting have become too complicated. I believe the patient stands the best chance with a timely operation. If the patient has had vomiting of blood prior to operation the diagnosis is relatively simple. All one needs to do at operation is to ascertain what the portal venous pressure is. If the portal pressure in a patient with massive hemorrhage who has had hematemesis is under 20 cm. of saline solution, one knows he is dealing with a hemorrhagic manifestation of the peptic ulcer diathesis such as an erosion or ulcer in the stomach or duodenum. If, on the contrary, the portal venous pressure is 40 cm. of saline solution or more, one knows he is dealing with portal hypertension in one of its manifestations. The acid-peptic factor is operative here too, I believe, owing to the exaggerated force of increased portal pressure. Herniation of a bit of turgid, erectile gastric mucosa occurs over the esophagogastric juncture. Owing to the great sensitivity of the esophageal mucosa to injury by acid peptic juice, esophageal erosion occurs with ensuant hemorrhage.

As Dr. Wirts pointed out, the whole category of bleeding from the alimentary tract must come into the purview when considering gastrointestinal hemorrhage. We are all too myopic to be able to resolve it correctly in every situation. Moreover, it is not always easy to decide what one should do.

I recall seeing a physician who came while on his vacation to talk with me about bleeding in the stool. I was inclined to believe he had a duodenal ulcer, though there had been no hematemesis. When another attack of hemorrhage and abdominal pain interrupted his vacation, I undertook to explore him. There was no duodenal ulcer, but a large perforated enterocystoma was found in the mid jejunum. We believed the tumor to be benign. About 10 years went by and this same physician died of a nasopharyngeal tumor with metastases to the liver. The patient's physician wrote me concerning the sections which checked closely with those found in the nasopharynx and liver. Ostensibly the enterocystoma, which had been called benign, was the primary lesion.

For the patient with a normal portal pressure who has had hematemesis, I do a segmental gastric resection and a pyloroplasty. These two procedures permit adequate inspection and examination of the duodenum, antrum and remaining fundus. If the portal pressure is increased, in years gone by I have usually done either a total gastrectomy or excision of the entire acid secreting area, anastomosing the antrum to the esophagus, after excision of the distal 4 to 5 cm. of esophagus, which segment in my experience almost invariably contains the erosive bleeding area. Experience has taught that, however, both total gastrectomy as well as the lesser procedure of total excision of the acid-secreting area—both are followed invariably by a pernicious anemia-like megaloblastic anemia in a period of 3 to 5 years. The intramuscular administration of Vitamin $B_{12}$ suffices to correct the anemia; however, this sequence suggests that some other therapeutic endeavor should be sought in patients having hemorrhage from a benign condition which would demand a near or total gastrectomy. It would appear that Dr. Merendino's interposition operation (*Ann. Surg.*, **142**:486-509, 1955) might be such a procedure. Excision of some of the upper fundus together with 4 to 6 cm. of distal esophagus should encompass the bleeding area in most instances of hemorrhage in portal hypertension. A Heineke-Mikulicz pylorotomy should be added, because the procedure would necessitate a bilateral truncal vagotomy. Whether esophageal varices may exist and give rise to hemorrhage in the absence of portal hypertension I do not know. It occurs in the rectum; perhaps it also occurs occasionally in the esophagus.

*Dr. Wirts:*—I would like to ask Dr. Wangensteen whether he has any plan of therapy that he uses for hereditary hemorrhagic telangiectasis with small bowel bleeding.

*Dr. Wangensteen:*—None.

# MASSIVE HEMORRHAGE OF THE UPPER GASTROINTESTINAL TRACT: INDICATIONS FOR SUBTOTAL GASTRECTOMY OR VAGECTOMY*†

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and

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In the past 28 years there have been 403 cases of massive upper gastrointestinal hemorrhage treated on the Fourth Surgical wards at Bellevue. This period of time has encompassed essentially six eras, each of which typifies a different therapeutic regimen, although the senior author has been more or less intimately associated with the conduct of these cases throughout all eras.

We have interested ourselves primarily in the immediate hospital mortality of massive bleeding of peptic origin when handled by three possible modes of treatment, and these data are summarized in chart form in Table I. The method of case selection is of some importance in the evaluation of such data; criteria for inclusion are therefore given in detail below.

1. The series contains no cases diagnosed as due to esophageal varices. We believe that it is almost always quite easy to distinguish this group, which constitutes an entirely different type of problem.

2. All cases found to be due to tumor, whether benign or malignant, have been eliminated from consideration in this paper.

3. Only those cases actually treated on the surgical wards of our service have been considered, thus omitting cases seen in consultation and not transferred.

4. While there have been minor changes through the years in the precise definition of what constitutes "massive" hemorrhage, the basic requirements for inclusion are: a) Admission hemoglobin 7.0 gm. or less and/or RBC 3.0 million or less; b) recent acute severe melena or hematemesis or both; and c) clinical hypertension with rapid pulse on admission, usually with concomitant signs of hypovolemic shock.

5. Within the above framework the reported cases are consecutive, and no case has been omitted from the series for other reasons.

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In the first era, from 1928 to 1937, 135 patients were treated, all "conservatively": that is, nonoperatively. There were 16 deaths, giving a mortality of 12 per cent. It appeared that among these fatal cases there might have been a considerable proportion salvageable by surgery, and accordingly in the period from 1937 to 1945 an effort was made to select and operate upon those who would otherwise have a fatal outcome. Under the direct supervision of one experienced surgeon this program was successfuly carried out, with a lowering of over all mortality to 5.6 per cent in 142 patients. Only one quarter (27 per cent) of the patients were subjected to surgery, 24 (17 per cent) as elective procedures after cessation of severe bleeding, and the other 14 (10 per cent) as emergency operations. In the former group there was 1 death (4 per cent), whereas in the latter there were 5 (35 per cent). If, however, we look upon the emergency group as having almost 100 per cent mortality under conservative therapy, we find that 9 of the 14 patients were salvaged. That this group was reasonably well selected is evidenced by the fact that there were only 2 deaths in the 104 patients conservatively treated.

The ensuing 3-year period shows a conservative reaction from the high operative mortality in emergency exploration; only one case was so treated, and survived. There was a 19 per cent mortality among the conservatively treated groups.

The fourth era reflects a different trend: less than half of the patients were discharged without operation. But the over all mortality rose even further, to 29 per cent, eight of the nine deaths occurring in the surgically treated group. We believe that this intolerable mortality points out the need for close supervision of the residents in such cases by one, or at most two, senior surgeons, whose delicacy of judgment and technic, based on years of experience with this specific problem, will enable the residents to choose, prepare, operate upon and carry through successfully this most difficult group of patients.

The past 4 years have shown an increase in the proportion of surgically treated patients to include almost ⅔ of the entire case load. Ten patients (22 per cent) underwent emergency surgery, with two deaths only, a not unsatisfactory outcome for this group. Of the 19 patients who had elective surgery, only one died; and only one death occurred in the nonoperative group, bringing our most recent over all mortality down to 9 per cent.

Our present attitude toward these patients is to attempt to handle them conservatively, using transfusions freely; but if the patient is bleeding faster than blood can conveniently be replaced, operation is indicated as an emergency. Subtotal gastrectomy with removal of the lesion is the only safe and proper procedure in the presence of a chronic, indurated ulcer; procedures of lesser extent too often fail to control the hemorrhage for which the operation was performed. We emphasize the close cooperation between medical and surgical teams at the resident level, with close supervision by experienced attending

TABLE I

MASSIVE HEMORRHAGE FROM PEPTIC ULCER
MORTALITY FOR VARIOUS PERIODS AND TYPES OF TREATMENT

| Period | Rx | No. of Pts. | % of Group | No. of Deaths | % of Deaths | Cases/month |
|---|---|---|---|---|---|---|
| 1928-37 | Non-op. | 135 | 100 | 16 | 12 | 1.22 |
| | Elect. op | 0 | 0 | 0 | 0 | |
| | Emerg. op. | 0 | 0 | 0 | 0 | |
| | Total | 135 | 100 | 16 | 12 | |
| 1937-45 | Non-op. | 104 | 73 | 2 | 2 | 1.35 |
| | Elect. op. | 24 | 17 | 1 | 4 | |
| | Emerg. op. | 14 | 10 | 5 | 35 | |
| | Total | 142 | 100 | 8 | 5.6 | |
| 1946-49 | Non-op. | 32 | 76 | 8 | 25 | .93 |
| | Elect. op. | 9 | 21 | 0 | 0 | |
| | Emerg. op. | 1 | 3 | 0 | 0 | |
| | Total | 42 | 100 | 8 | 19 | |
| 1949-52 | Non-op. | 14 | 44 | 1 | 7 | .89 |
| | Elect. op. | 9 | 28 | 4 | 44 | |
| | Emerg. op. | 9 | 28 | 4 | 44 | |
| | Total | 32 | 100 | 9 | 29 | |
| 1952-56 | Non-op. | 16 | 36 | 1 | 6.2 | 1.00 |
| | Elect. op. | 19 | 42 | 1 | 5.4 | |
| | Emerg. op. | 10 | 22 | 2 | 20 | |
| | Total | 45 | 100 | 4 | 9 | |
| (Emergency op.: no ulcer) | | (7) | | (2) | (29) | |

| Period | Rx | No. of Pts. | % of Group | No. of Deaths | % of Deaths | Cases/month |
|---|---|---|---|---|---|---|
| 1928-56 | Non-op. | 301 | 76 | 28 | 9.3 | 1.16 |
| | Elect. op. | 61 | 15.4 | 6 | 9.8 | |
| | Emerg. op. | 34 | 8.6 | 11 | 32.4 | |
| | Total | 396 | 100 | 45 | 11.4 | |
| Total including emergency operations for ulcer | | 403 | | 47 | 11.7 | |

men. We feel that elective surgery after control of hemorrhage is frequently indicated, particularly in the older age groups, in those who have bled massively before, and in patients who have already demonstrated intractability to medical management.

Taking the entire 28-year period as a whole, we find that about three-quarters of the case load was handled without operation and had a mortality of 9.3 per cent. One sixth of the patients had elective surgery after cessation of bleeding, with 90.2 per cent survival. Less than one tenth (8.6 per cent) underwent emergency operation; the mortality of this group was 32.4 per cent. The over all mortality, omitting patients explored and found to have no ulcer, is 11.4 per cent.

In the most recent 4½-year period, seven patients underwent laparotomy for uncontrollable upper gastrointestinal bleeding and at the time of laparotomy no source of the bleeding could be found. Two of this group died despite subtotal

TABLE II

TOTAL NUMBER OF PATIENTS OPERATED UPON FOR PEPTIC ULCER OR HEMORRHAGIC DIATHESIS FROM JANUARY 1952 TO JULY 1956

| Diagnosis | Elective | Emergency | Total |
|---|---|---|---|
| Duodenal ulcer | 115 | 2 | 117 |
| Gastric ulcer | 19 | 0 | 19 |
| Gastrojejunal ulcer | 4 | 1 | 5 |
| Hemorrhagic diathesis | 4 | 5 | 9 |
| | 142 | 8 | 150 |

gastric resection, bringing the mortality of the entire series of 403 cases to 11.7 per cent.

Apropos of this latter group, Table II shows the experience of the senior author in his private practice during the same 4½-year period. Of 8 cases requiring emergency surgery for uncontrollable bleeding, only 3 were demonstrably due to frank ulcer. Pooling this information with the above, it is seen that in the combined experience, ward and private, of 25 emergency explorations for massive gastroduodenal hemorrhage, 12 were found to have no ulcer at operation. This demonstrates not only the virtual impossibility of identifying these bleeders preoperatively, but also the relative frequency with which the problem has arisen in recent years.

Table III presents the data on the 5 private cases operated as emergencies, along with 4 others which were operated as elective cases and in whom, likewise, no frank ulcer was found. All 9 of this latter group received subtotal

TABLE III

HEMORRHAGIC DIATHESIS—EMERGENCY OPERATION

| Case No. | Sex | Age | Melena | Hematemesis | Previous Bleeding | RBC. Adm. (M) | Hgb. Adm. (gm.) | Preop. Transf. (c.c.) | Op. Transf. (c.c.) | Postop. Bleeding | Follow-up (months) | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 43 | + | 0 | 3X | 2.6 | 7.5 | 59,000 | 1,500 | 0 | 45 | Bled after Stg for Du |
| 2 | M | 61 | + | + | 1X | 3.0 | 8.0 | 5,000 | 1,000 | 0 | 37 | Neg. GI series preop. |
| 3 | M | 75 | + | 0 | 0 | 2.5 | 7.0 | 7,500 | 3,000 | 0 | 23 | Cortisone Rx |
| 4 | F | 50 | + | + | 1X | 3.0 | 7.0 | 3,500 | 2,000 | 0 | 16 | Previous Pge |
| 5 | M | 71 | + | + | 0 | 3.3 | 7.5 | 2,500 | 1,500 | 0 | 11 | Cirrhotic without varices |

HEMORRHAGIC DIATHESIS—ELECTIVE OPERATION

| Case No. | Sex | Age | Melena | Hematemesis | Previous Bleeding | RBC. Adm. (M) | Hgb. Adm. (gm.) | Preop. Transf. (c.c.) | Op. Transf. (c.c.) | Postop. Bleeding | Follow-up (months) | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 6 | M | 31 | + | 0 | 2X in 3 months | 5.0 | 15.0 | 2,500 | 0 | 0 | 43 | X-rays reported duod. ulcer |
| 7 | M | 63 | + | 0 | 3X in 2 years | 4.5 | 11.5 | 1,000 | 1,500 | 1X | 24 | 3 x-rays non-diagnostic |
| 8 | F | 58 | + | 0 | 4 years | 3.65 | 9.0 | 1,500 | 0 | 0 | 30 | X-rays "positive" for Du |
| 9 | M | 71 | + | 0 | 2 months | 3.0 | 8.0 | 0 | 0 | 0 | 10 | Suspected gastric malig. |

vagectomy as their definitive surgical procedure, with survival of all cases. Only one bled subsequent to this procedure, and this was a minor episode which ceased quickly on conservative treatment.

It is our feeling that this experience suggests an alternative course to the "blind" gastrectomy widely advocated under such circumstances at the present time. A summary review of 34 articles in the past 5 years on this subject reveals that half the writers recommend wide subtotal gastrectomy, while only ¼ explicitly condemn it. Granted that the experience of the operator justifies his acceptance of palpation and inspection as negative for frank ulcer, it is reasonable to suppose that morbidity and mortality will be lower if the gastrointestinal tract is not opened. And if, as this experience appears to indicate, the hemorrhage can be controlled by vagus resection, the procedure deserves a definite place in the armamentarium of the surgeon confronted by massive hemorrhage from the upper gastrointestinal tract.

## Discussion

*Dr. O. H. Wangensteen:*—Dr. Snapper and I have been debating for years how properly to treat a patient with massive hemorrhage. It would be good to know whether Dr. Hinton has been able to delineate a program by which he could fit cases into categories for treatment.

Perhaps I ought first to say something about so-called empiric gastric resections, for the origination of which procedure I hold some responsibility. I did an empiric gastric resection for massive gastric hematemesis first in 1944 (*Canad. M. A. J.* **53**:309, 1945). The most frequent finding in such cases is a small, superficial erosion high up on the lesser curvature of the stomach and slightly posterior.

In more recent years, I have found when operating for massive gastric hemorrhage accompanied by hematemesis and no lesion is found at operation when the stomach is examined from the outside that, segmental gastric resection affords a satisfactory mode of attack upon such problems. If no lesion can be seen or felt, I make a rather long pylorotomy incision which provides a satisfactory means of determining whether the bleeding originated in the duodenum or the antrum. If there is a duodenal erosion or ulcer, the bleeding point is dealt with. If a crater is present, the mucosal edges are approximated over the crater after the bleeding is arrested with a stitch. The pylorotomy incision then is converted to a Heineke-Mikulicz pyloroplasty with transverse closure of the longitudinal incision in the stomach and duodenum.

If the bleeding point is found in the antrum, a Billroth procedure is done. If the lesion is a duodenal ulcer, a segmental resection is done. Similarly, if no lesion is found in the antrum, the midgastric segment is excised, opened and examined for a source of bleeding. If no source for the hemorrhage is observed

in the excised specimen, the more proximal residual gastric pouch is examined. This is not difficult to do. When no lesion is found in the duodenum, antrum, excised midgastric segment or proximal residual gastric fragment, the usual finding in such instances of occult massive hematemesis has been a diffuse microscopic ulcerative gastritis.

Spencer and Maloney (*Surgery*, **40**:904, 1956) favor a 7-inch long gastrotomy incision to search for the bleeding point in instances of massive hemorrhage. If no bleeding is encountered, they terminate the operation by merely closing the incision. If a bleeding point is encountered, it is dealt with by conventional technics. Where, however, no bleeding point is seen, I prefer to complete the operation in massive gastric hemorrhage attended by hematemesis by doing a gastric resection.

It is always important to measure the portal venous pressure in all cases operated upon for massive hemorrhage to exclude the possibility of portal hypertension.

It is apparent that Dr. Hinton has not abandoned employment of transfusion in the management of massive hemorrhage, for he mentioned five patients with uncontrolled hemorrhage, whom he was able to rescue by transfusion and well-timed surgery. Moreover, Dr. Hinton it would appear, holds the world's record for large transfusions in massive hemorrhage. Ten years ago I did a total gastrectomy in a boy of 17 years for massive recurrent gastric hemorrhage owing to extrahepatic portal hypertension. It was necessary to give 65 transfusions in order to get the patient in satisfactory condition for an emergency gastrectomy. That young man is well and has completed a course in dentistry and practices dentistry in our area. It is interesting that this patient was not even jaundiced following all these transfusions. Even in 1956 it is not easy to transfuse a patient 65 times without incurring jaundice.

There probably can never be a final and definitive answer to how all patients with massive hemorrhage should be treated. It is obvious from what Dr. Hinton has told us that ordinarily he is wont to take a less aggressive course in the management of massive hemorrhage than some of us do.

Dr. James Casey of our group studied the deaths from peptic ulcer reported in the vital statistics of 1952 for the State of Minnesota [*Minnesota Med.* **37**:448, (June), 1954]. There were 129 deaths and of these, Dr. Casey found that hemorrhage was the sole cause of death in 46 per cent of the cases. In an additional 5 per cent hemorrhage had occurred with perforation. In other words, the foremost cause for the continuing large mortality from ulcer today is hemorrhage. We need to implement measures to assure a more secure and definitive control of the ulcer problem. Too many patients with peptic ulcer controlled by conservative medical treatment end up in the mortality column from hemorrhage or perforation.

These discussions of a difficult problem, though necessarily somewhat controversial are nevertheless very timely. It probably will be a long time before there can be more uniform agreement on how to deal with the difficult problem of massive gastric hemorrhage. For myself, may I say that I find no difficulty in defending gastric section more ardently than Dr. Hinton does in the control of massive gastric hemorrhage. However, he speaks from a large experience critically evaluated and it is important that we be attentive to his opinion and examine carefully the evidence which he has presented to support it.

*Dr. I. Snapper:*—Dr. Hinton has clearly elucidated how much more serious are the cases of apoplexia of the stomach than of hemorrhage of a typical gastric or duodenal ulcer.

The figures which Dr. Hinton has given are highly interesting because they started in the year 1928. This is the crucial year when blood transfusions became popular and again one obtains the impression that the more the patients are transfused, the higher the mortality

The administrations of the special teams which take care of the upper gastrointestinal hemorrhages by transfusing the patients until the hemoglobin is raised to 10 gm. per cent and the blood pressure to 120 mm., are not conducive to reducing the fatality of the patients they administer to,—on the contrary.

When blood transfusions are used in moderation—just to keep the patients with gastric hemorrhage out of shock—the over all fatality will come down to 2-3 per cent, the pre-1928 figure, and the necessity of a surgical intervention for hemorrhage due to peptic ulcer will only rarely arise.

*Dr. Mixter:*—I trust you will excuse me if I explain that it is very easy for the senior to talk about his junior's paper, because policy may be changed thereafter. In my case, it is quite the reverse and I have to be a little bit careful. (Laughter)

Dr. Wangensteen asked if Dr. Hinton might tell in a little more detail how the patients were selected for surgery, and perhaps more specifically, how they were selected for vagectomy. This, of course, involves not only a matter of definitions but also of very fine surgical judgment, and in the final analysis, as Dr. Hinton pointed out in his talk, the matter should be in the hands of one, or perhaps two people.

Essentially the definition of massive gastrointestinal bleeding has been made on a clinical basis, aided only by the most primitive types of laboratory study. One can admire, perhaps, at a distance, Dr. John Stewart's detailed examination of blood volume, and various others', but in our hands a red count below 4,000,000, and hemoglobin below 7 acutely acquired in the presence of evidence of active bleeding, either by gastric tube or by tarry stool, is sufficient to call the patient a massive bleeder. None of these patients has had a normotensive

level. There were some hypertensives who carried a systolic around 100 or 110, but these were persons who normally ran up to 200. Many of them, of course, in the series that was discussed, had shock levels at the time they were admitted. These are the patients about whom we were speaking.

Surgically, the present attitude is that all patients should be under the observation of a surgeon from the moment they enter the hospital; and if they are on the medical ward temporarily, that they should be under the care of a surgeon who sees them throughout.

If a patient enters in shock, his physiology should be restored by transfusion, and a large needle should be used, so that in the event of exsanguinating hemorrhage, the blood pressure may be maintained. If a patient, with eight or ten hours of massive transfusion—and by this I mean 4,000 to 5,000 c.c. of blood —is unable to maintain blood pressure and a low pulse, he is considered a surgical candidate. One must specifically omit those few patients in whom one is never able to bring the blood pressure up to operable levels. We lost one five or six weeks ago in whom we were not able to bring the pulse below 140, or the blood pressure above 70 at any time. This patient expired, and at autopsy was found to have a callous ulcer of the duodenum with a branch of pancreaticoduodenal wide open at the bottom.

I can speak very strongly for Dr. Hinton in making clear the distinction between the patient with the callous ulcer with a bleeding vessel, and the patient who is bleeding from the surface of an abnormal stomach. There is no question in anyone's mind as to what should be done with the former. It is the latter group in whom we are interested, and who have responded in each case, to vagectomy.

*Dr. Wangensteen:*—Do you do any auxiliary surgery like drainage operation?

*Dr. Mixter:*—The question has been submitted to me: Do you perform vagectomy alone without any anastomosis? Yes. He also asks, here: Do you open up the stomach for inspection?

No. The procedure of subtotal gastrectomy with removal of the ulcer is a curative one, if feasible, in the presence of a callous ulcer with a bleeding vessel. If the ulcer is not callous, it is almost impossible that there should be a large bleeding vessel at the bottom of it. Dr. Hinton feels that by palpation one can tell all one needs. This, of course, is by a skilled hand.

There is a question by Dr. Eisenstadt: Is there any case in which the spleen was ruptured and a splenectomy had to be done? No.

Dr. Bruger asks: How can you be sure there is no definite lesion present in a patient who is having gastrointestinal bleeding? Wouldn't the blood in the stomach and/or duodenum interfere with x-ray or other attempts to arrive at a diagnosis?

I might say here that we have never hesitated to submit the patient to emergency radiologic study, and although we have no figures to present, I should estimate that somewhere in the neighborhood of 25 per cent of such active bleeders would give sufficient evidence to give one a working diagnosis by x-ray even in the presence of blood. If the x-ray is not diagnostic, one proceeds as the airplane pilot does without instruments, flying by the seat of his pants.

Here is an unsigned question: I have seen no analysis of mortality from gastrointestinal hemorrhage from the viewpoint of poor surgical risk. Since these patients as a group appear to account for a major portion of surgical deaths, apparently because they cannot withstand anoxia, what is thought of immediate gastrectomy in such cases, without regard to diagnosis or trial medical therapy?

I think this question has been largely answered. If one divides the bleeders into those who are controllable and those who are uncontrollable, then the controllable group either arrives at medical therapy, or, in the presence of specific indications for surgery—which I believe we ought not go into—receives an elective gastrectomy. The uncontrollable bleeders are divided into those who have a callous ulcer, either by history or x-ray examination: these persons should naturally have a subtotal gastrectomy; those bleeding from unknown cause must, if the bleeding turns out to continue uncontrollable, be explored, and if that exploration results in no palpable ulcer being found, we believe these patients should receive vagectomy.

If a callous ulcer is found at this time, naturally a subtotal should be performed.

Are there any other questions?

*Dr. Snapper:*—Are there any statistics prior to 1928, that is, statistics, for instance, of nonoperated people, as from 1928 to 1937?

*Dr. Mixter:*—Dr. Snapper asks if there are any statistics prior to 1928. I regret that there are none, and the only reason that there are statistics in this particular instance is due to Dr. Hinton's belief in the necessity for doing as that famous bird did, flying backwards and seeing where you have been.

*Dr. Snapper:*—I wish we could slide backwards a little bit more.

*Dr. Mixter:*—As some of you may know, the records at Bellevue frequently do not lend themselves to an accurate and exhaustive analysis. (Laughter)

*Dr. Mixter:*—Dr. Ditmore, from Boston, asks what has been our experience with balloons and tubes. In the presence of bleeding from esophageal varices, a tube of the Sengstaken or Blakemore type ought to control bleeding if properly applied. I believe there should be complete agreement on this subject. This, in fact, forms an excellent diagnostic test. Those patients who bleed from varices do not at any time enter into the discussion which Dr. Hinton made.

So far as gastric tubes are concerned, in the presence of acute bleeding, we have no fear of them. Almost all of our bleeders have a tube put down, primarily at first for diagnosis, and later to watch the course of possible future hemorrhage.

*Question:*—I should like to know what percentage of patients vagectomized came to autopsy, and what percentage of those autopsies have shown ulcers not found previously.

*Dr. Mixter:*—In this group of nine patients presented this morning, plus three done in the last few months, there were no deaths, consequently, no autopsies. We had one case which came under our observation but not under our conduct, in whom a known duodenal ulcer was present. The resident surgeon remarked at the time that that ulcer, which was anterior, in the first portion of the duodenum, hardly seemed to account for the patient's severe bleeding. A subtotal gastrectomy, comprising approximately 70 per cent, was done. The patient bled postoperatively, and the specimen showed gastritis. The patient exsanguinated, came to autopsy, and was found to have extensive superficial ulceration in the region of the cardia.

## NEWS NOTES

### World Congress of Gastroenterology

The World Congress of Gastroenterology will convene at the Sheraton-Park Hotel, Washington, D.C., 25-31 May 1958. Guest speakers from many nations will participate, as well as members of the profession from the United States.

The program will be divided into five major categories: 1. Peptic Ulcer. 2. Carcinoma of the Stomach. 3. Malabsorption and Sprue-like States. 4. Nutrition in Relation to Lesions of the Liver and Pancreas. 5. Infections and Infestations of the Alimentary Tract. Emphasis will also be put on training of the postgraduate student in diseases of the digestive tract. Modern methods of translation will enable each person attending the Congress to hear the presentations in the language of his choice.

Commercial and scientific exhibits will be held in conjunction with the Congress in the Sheraton-Park Hotel.

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### Twenty-Fifth Anniversary Dinner

The New York Academy of Gastroenterology, chapter of the American College of Gastroenterology, held its annual dinner meeting at the Hotel Delmonico in New York City. This affair marked the twenty-fifth anniversary of the chapter which was the original Society for the Advancement of Gastroenterology, forerunner of the present American College of Gastroenterology.

Appropriate recognition of the occasion was made in the address of Dr. A. Xerxes Rossien, outgoing President of the Academy. Dr. Rossien traced the history of the organization from its start at the home of Dr. Samuel Weiss in 1932 through the present day.

Plaques were presented to Dr. Rossien and two other past Presidents of the Academy, Drs. Harry Barowsky and Franz J. Lust for their services.

At the annual meeting officers of the Academy elected for 1957-1958 were: President, Dr. Jerome A. Marks; President-elect, Dr. Milton J. Matzner; 1st Vice-President, Dr. Michael Weingarten; 2nd Vice-President, Dr. Joseph R. Van Dyne; Secretary, Dr. Alexander Slanger; Recording Secretary, Dr. Jerome Weiss; Treasurer, Dr. William C. Jacobson and Chairman, Board of Trustees, Dr. Harry Barowsky.

## *President's Message*

### The Road Ahead

The American College of Gastroenterology is in an enviable position of leadership. With its pleomorphic membership, embodying the grass roots general practitioners, and rising upwards to the Fellowship boarded in the allied specialities, it can develop an important place for gastroenterology in medicine.

The College can and must assume leadership in the stimulation of research work by young men in the field. There is definitely a need for researchers in our speciality. The reason we lack importance as a speciality, lies with the need for public relations and stimulation of interest in gastroenterology. This requires the development of interested teachers in basic sciences of biochemistry, pathology and physiology of gastroenterology. At present, teaching in this field is lackadaisical and teaching facilities are sadly lacking. We must develop research funds in order to develop the best teaching facilities possible.

There is a need for development of public and professional interest. Consideration should be given to the development of a major national philanthropic project for peptic ulcer and hepatitis research. Many other organizations have been successful in developing their proper place in medicine by such a program. To be effective, however, all organizations in gastroenterology should work together. This spirit of cooperation between the various gastroenterological groups is essential in all the fields.

The "live and let live" philosophy should be adopted. One obvious place of orderly cooperation lies in the section of Gastroenterology and Proctology of the American Medical Association. The American College is particularly qualified for such participation because the American Medical Association is primarily a general practice organization and we give recognition to general practitioners with a special interest in gastroenterology. Yes, our speciality can gain the prestige it should have by being geared to the idea of general unity. It is the responsibility of the College to assume the leadership in developing the Road Ahead.

Arthur A. Kirchner

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## GASTROINTESTINAL TRACT

### PANEL DISCUSSION ON THE COMPLICATIONS OF PEPTIC ULCER: SURGERY FOR PEPTIC ULCER OF THE DUODENUM: Deryl Hart, Robert L. A. Keeley, W. Glenn Young, Jr. and William G. Anlyan. North Carolina M. J., 17:197 (May), 1956.

Since the hazards of gastric resection have been greatly reduced in the last two decades, the authors make a plea for earlier evaluation of peptic ulcer and the earlier referral of the more virulent group for surgery. In the absence of complications, however, a reasonable trial of medical therapy should be given in all cases. Sympathectomy and vagus nerve resection are not recommended because of high mortality and morbidity resulting from the procedure.

Partial gastrectomy (resection of 60-65 per cent of the stomach) is preferred to subtotal gastrectomy, since running the risk of a few marginal ulcers in the former procedure is preferable to the creation of a large number of nutritional cripples in the latter more drastic resection.

One of the more serious complications of gastric resection is duodenal fistula which occurred in 14 out of 248 cases reported. It is felt that a number of these complications might have been avoided had the patient's condition been evaluated and surgery advised at an earlier stage.

A. J. BRENNER

### PANEL DISCUSSION ON THE COMPLICATIONS OF PEPTIC ULCER: PERFORATED PEPTIC ULCER: George H. Wadsworth. North Carolina M. J., 17:201 (May), 1956.

Fifty per cent of peptic ulcers form and heal without ever being seen by a physician. Some perforations will occur as the first symptom of the disease. Aggravating factors preceding perforation are vague. There seems to be no relationship of the onset of perforation to meals or activity. Fatigue and severe emotional upset are, however, common before perforation. Perforations are 3 times more common in men, most of them occurring on the anterior wall of the duodenum. Diagnosis is made easy when there is severe sudden diffuse and more or less constant epigastric pain radiat-

ing to the shoulders. In 50 per cent of cases muscle spasm and tenderness over the entire abdomen is always present and rebound tenderness is rarely absent. Free air under the diaphragm is present in ¾ of the cases.

According to the authors simple closure of the perforation is still the method of choice. In cases of doubtful diagnosis, in poor risk cases and in those where the interval after perforation is greater than 24 hours, conservative therapy with continuous suction may be tried. Immediate gastric resection has been tried in Europe and advocated by some in the U. S.

A. J. BRENNER

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**PANEL DISCUSSION ON THE COMPLICATIONS OF PEPTIC ULCER: PEPTIC ULCER OBSTRUCTION: Hubert C. Patterson. North Carolina M. J., 17:203 (May), 1956.**

Obstruction is the most common complication of peptic ulcer and oft times the first indication of the disease. It is present in 25 per cent of surgically treated cases. The degree of obstruction is generally not relative to the severity of the symptoms. The stenosis present is due to some cicatrization and to a great extent also to varying degree of spasm and edema. The edema is probably the precipitating cause of complete obstruction. Because of starvation these patients are usually dehydrated and poor surgical risks. They should be brought into electrolyte and nutritional balance and operated on at the earliest favorable time. Billroth I procedure with vagotomy is recommended by the authors for the best postoperative results.

A. J. BRENNER

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**PANEL DISCUSSION ON THE COMPLICATIONS OF PEPTIC ULCER: MALIGNANT CHANGE IN PEPTIC ULCER. Hubert C. Patterson. North Carolina M. J., 17:205 (May), 1956.**

The possibility of malignant change in a duodenal ulcer must be considered rare. Malignant degeneration in chronic gastric ulcer, however, occurs in about 5-10 per cent of cases. The authors find it difficult to believe that carcinoma is the primary lesion in long standing gastric ulcers. The diagnosis of carcinoma in the fungating projecting type which never penetrates the muscle wall is easily made. The penetrating small carcinoma with peptic ulceration, however, is the type that gives us trouble in diagnosis. According to Jordan 50 per cent of ulcers in the cardia are malignant, 60 per cent of ulcers on the greater curvature are malignant. Every available diagnostic procedure should be utilized to rule out malignancy in peptic ulcer cases. Anacidity is definitely in favor of malignancy. If an ulcer persists or recurs, it is probably cancer. Lesions must completely disappear and not recur roentgenologically to be considered benign. Since ¾ of gastric ulcers are probably malignant and many cases of those thought to be benign are found to be malignant after surgery and also since 40-50 per cent five-year cure rates can be obtained in the latter group, surgery is recommended in all cases where medical care has failed and in those where the diagnosis is in doubt.

A. J. BRENNER

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**PANEL DISCUSSION ON THE COMPLICATIONS OF PEPTIC ULCER: MASSIVE HEMORRHAGE IN PEPTIC ULCER: W. W. Shingleton, W. G. Anlyan, R. L. Keeley, W. G. Young and Deryl Hart. North Carolina M. J., 17:207 (May), 1956.**

Since the treatment for massive hemorrhage in peptic ulcer is still not standardized, all such patients treated at Duke Hospital between 1930 and 1953 have been analyzed. Ten per cent of the population of the U. S. at some time in their lives suffer from peptic ulcer, 10-20 per cent of these experience gross hemorrhage. All cases were admitted to the medical service and treated with diet, supportive medications and massive transfusions for 24 to 36 hours. If there was no improvement, as judged by blood pressure, pulse and hematocrit, early operation was performed. Age

of patient and number of previous episodes were not given much consideration. There was definitely a higher mortality in patients with severe hemorrhage operated on after 48 hours from the onset of bleeding. It is believed that liver anoxia causes irreversible shock in prolonged hemorrhage. Surgery after the bleeding has stopped, depends on many factors including age, presence of hypertension and number of previous episodes of bleeding.

A. J. BRENNER

## INTESTINES

**THE MANAGEMENT OF ACUTE AND CHRONIC INTESTINAL DISEASES: Philip W. Brown. North Carolina M. J. 1751 (Feb.), 1956.**

The causes of belly pain and diarrhea, singly or in combination are so numerous and not necessarily primary in the intestinal tract. For this and other reasons the author has limited his discussion to 4 groups of infections, usually acute diseases of the bowel and 3 due to organic lesions, usually presenting a chronic picture or an acutely recurring one. Viral enterocolitis, salmonellitis, and those traceable to the shigella and staphylococcus groups are discussed as well as diverticula of the colon, ulcerative colitis and regional enteritis (realizing that there is a group that fits a combination of the latter two).

However brief the discussion of each of these diseases appears, the available practicable knowledge of the complications and treatment of these is well summarized. There is very little to disagree with in the article except that amebiasis should not have been passed off as being less common than the staphylococci infections. The manifestations and complexities of the *Endameba histolytica* span over both the acute and chronic intestinal diseases. Diagnostic procedures are far from simple and the incidence is far from low.

A. M. SUSINNO

**PARASITIC DIARRHEA: John D. Stull. Illinois M. J. 109:119 (Mar.), 1956.**

Parasitic bowel infestation in children is relatively common and failure to diagnose these conditions is equally common. Needless to say, the clinician must be suspicious in all cases because of the vague and at times bizarre symptomatology.

The diagnosis of *E. Histolytica* and other parasites depends on a well-staffed, interested laboratory and numerous stool examinations. The stools obtained fresh for demonstration of the motile forms should be supplemented by the zinc-sulfate flotation method for concentration of cysts.

A discussion of the various drug medications is included.

IRVIN DEUTSCH

**CLINICAL TRIAL OF GLAUCARUBIN IN TREATMENT OF AMEBIASIS: E. C. Del Pozo and M. Alcaraz. Am. J. Med. 20:412 (March), 1956.**

Seventy-eight patients with chronic amebiasis (cysts found in stools) and nine patients with amebic dysentery (vegetative forms noted) were treated with Glaucarubin, a crystalline glycoside derived from simarouba. The patients were studied in six different groups. The daily dose varied from 10 to 280 mg. and the duration of treatment varied from 5 to 32 consecutive days. Clinical improvement was evident from the first to the sixth day after treatment had begun. No toxic symptoms were noted and the drug was well tolerated. Negative stools were reported as early as the first day of treatment and in only two patients did the parasites persist. After treatment was terminated 54 patients were observed for a period of 1 to 13 months. Eight of the patients with chronic amebiasis developed recurrence of *E. histolytica*.

JOHN M. MCMAHON

**CAUSE AND CARE OF EXTENSIVE RECTAL FISTULAS: Walter A. Fansler. Texas J. Med. 52:237 (Apr.), 1956.**

The three causes for extensive rectal fistulas are 1. perforation of the rectal wall above the "sphincter mechanism" by trauma, foreign body, infection or neoplasia 2. extensive neglected peri- and pararectal abscesses 3. improper drainage of supralevator abscess through the ischiorectal fossa rather than through the rectal wall.

The treatment of peri- or pararectal abscesses is early incision and drainage even before skin changes are seen.

Fistulas with the internal opening above the "sphincter mechanism" often require division of a sufficient portion of "sphincter mechanism" to produce incontinence.

Where there is extensive soft tissue damage involving one or both ischiorectal fossae, it is necessary to produce a wound that is flat and broad so that healing will occur from the bottom up without bridging.

ALEXANDER ZABIN

## LIVER AND BILIARY TRACT

**ACUTE CHOLECYSTITIS OF CHILDHOOD: James A. Webb. J. Michigan M. Soc. 55:431 (Apr.), 1956.**

Acute cholecystitis in children is an uncommon disease. Only about 30 cases have been reported since 1948. Even though the incidence of the disease is very low, the treatment assumes great importance, especially when the diagnosis is made at the operating table. The preoperative diagnosis on the 7-year old male patient described in the case report was acute appendicitis. When the peritoneum was opened, free fluid was found and the appendix had prominent vascular markings. However, these findings were not of sufficient magnitude to explain the symptomatology and leucocytosis. Further exploration revealed a thin-walled gallbladder with a gangrenous fundus and without stones. The microscopic diagnosis was acute and chronic cholecystitis, and normal appendix. The importance of further exploration within the peritoneal cavity when the initial findings do not unequivocally support the preoperative diagnosis is stressed.

CHESTER S. SVIGALS

**OPERATIVE CHOLANGIOGRAPHY: Royal A. Weir and Carlos Lizama. Missouri Med. 53:280 (Apr.), 1956.**

Operative cholangiography is visualization of the biliary structures obtained by injection of an opaque medium directly into the main biliary ducts during any surgery upon the biliary tract. The authors maintain that, if done routinely, this procedure makes exploration of the ducts easier and more complete, lowers the risk of leaving stones behind, and increases the opportunity of discovering pathology. They feel that morbidity and postoperative mortality are reduced by this procedure, as indicated in a series of 83 cases out of 232 cholecystectomies. Two technics are described, with advantages and disadvantages of each. The necessity for accuracy of technic is stressed, since minimal errors may produce false positive or false negative results.

The authors suggest operative cholangiography as a routine procedure.

JOHN N. DILL

**INTERNAL BILIARY FISTULA: Elias N. Tsardakas and Ausey H. Robnett. Cleveland Clin. Quart. 23:124 (Apr.), 1956.**

Internal biliary fistula is an abnormal communication between a portion of the extrahepatic biliary tract and any other viscus. In operative series it occurs in from 0.5 to almost 1 per cent. The authors report 18 cases. In 90 per cent of cases a gallstone is the provocative agent, 6 per cent a perforating ulcer; in 4 per cent carcinoma, abscess, or some rare condition such as echinococcus cysts. The most common form is a communication between the gallbladder and duodenum (cholecystoduodenal), next

cholecystocolic and choledochoduodenal.

The diagnosis depends on x-ray; thus in the 18 cases presented by the authors barium was present in the biliary tract in 10; and air in 5 cases; very often the actual fistulous tract can be delineated by the roentgenologist.

In the presence of symptoms the treatment is surgical. The usual treatment is cholecystectomy and closure of the duodenal or colonic opening. In the type due to duodenal ulcer the authors prefer subtotal gastrectomy and vagotomy and gastrojejunostomy, which they state promotes healing of the ulcer and protects the biliary tract from reflux of gastrointestinal contents.

SAMUEL L. IMMERMAN

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**SERUM IRON IN THE DIAGNOSIS OF HEPATOBILIARY DISEASE: L. Schamroth, W. Edelstein, W. M. Politzer and N. Stevens. Brit. M. J. 4973:980 (28 Apr.), 1956.**

The authors attempt to differentiate between obstructive and nonobstructive jaundice by determining that serum iron levels are higher in nonobstructive and normal in obstructive jaundice.

The normal serum iron level was set at 70-185 mcg. per 100 c.c.; above 220 was regarded as high. They then tabulated a series of 67 cases where serum iron levels were done 104 times. In the majority of the virus hepatitis it was never above 220 and in nearly all cases of cirrhosis of the liver and extrahepatic obstruction it was below 220.

There is no relationship between serum iron levels, serum bilirubin and severity of disease. The exception to a high serum iron level above 220 may be a patient with hemachromatosis developing an extrahepatic obstruction or cirrhosis of the liver. Hence the authors feel this test should be evaluated in conjunction with the clinical assessment of the patient.

LOUIS K. MORGANSTEIN

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**DIVERTICULUM OF THE GALLBLADDER: Thomas K. Rathmell, Samuel J. Lloyd and Raymond A. McCormack. Pennsylvania M. J. 59:670 (June), 1956.**

This case report deals with a diverticulum of the gallbladder. There is no distinctive symptomatology associated with this condition which is a pathologic entity. The authors point out that by the addition of their criteria, "grossly apparent ostia and encompassment by continuous bundles of smooth musculature, distinctive from the general wall of the gallbladder," many of the so-called diverticula are in reality Rokitansky-Aschoff sinuses.

IRVIN DEUTSCH

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**THE PROBLEM OF CHRONIC LIVER DISEASE IN YOUNG WOMEN: A. G. Bearn, H. G. Kunkel, and R. J. Slater. Am. J. Med. 21:3 (July), 1956.**

The authors have studied a total of 26 cases of hepatic cirrhosis occurring in young women predominantly. These were considered together because they exhibited certain clinical and biochemical characteristics which were not ordinarily seen in cases of either classic, posthepatitis or Laennec's cirrhosis. Certain features noted included arthritis, obscure febrile episodes and occasionally hormonal disturbances. The sex incidence, the generalized nature of the disease in certain persons, involvement of joints, pericardium and lungs as well as liver raises the possibility that some other disorder, possibly related to the collagen diseases, may play an important part in the disease process in some instances. Typical L. E. cells were noted in the blood of one patient and striking improvement followed the use of cortisone in some cases. Laboratory investigations in the acute stages of the disease, usually revealed an extremely high serum gamma globulin and an increase in the plasma cells of the liver. Although many of the signs and symptoms of classic *lupus erythematosus* were lacking in these patients and cirrhosis is a rare finding in the latter disease. The possibility that specific endocrine influences, present in young women, modify the usual course of infectious hepatitis is discussed. In most of the patients, the etiology of the disease process was uncertain.

JOHN M. MCMAHON

## BOOK REVIEWS FOR GASTROENTEROLOGISTS

**DISEASES AND DISORDERS OF THE COLON: Anthony Bassler, M.D., F.A.C.P., F.A.C.G., LL.D. 217 pages, profusely illustrated. Charles C. Thomas, Springfield, Ill., 1957. Price $6.75.**

Dr. Bassler needs no introduction to the medical profession. He has been in the field of gastroenterology over 50 years and has written numerous texts on this specialty. Dr. Bassler is also a contributor to many journals in this country, as well as in other countries. He is the dean of gastroenterology and has served in various capacities in numerous medical organizations, including that of past president of the American College of Gastroenterology.

Diseases and Disorders of the Colon summarizes all the latest medical, surgical, laboratory and diagnostic fields including treatment.

The general practitioner, radiologist and gastroenterologist will find useful information in this monograph. It is highly recommended.


Relax the best way
... pause for Coke

continuous quality
is quality you trust

DRINK
Coca-Cola

*"...an ideal treatment for the diarrheal syndrome..."*[1]

# RESION

(POLYAMINE METHYLENE RESIN AND SYNTHETIC SILICATES)

## *faster relief*[2]

In 90 patients treated with Resion, 86 (95%) were controlled in 8 to 12 hours, even faster than with bismuth and paregoric.

## *twice as effective*[2]

| THERAPY | % SUCCESSES |
|---|---|
| **RESION** | **92** |
| Kaolin and Pectin | 40 |
| Bismuth and Paregoric | 50 |

## *safe*[3]*...and non-constipating*[1-3]

The multiple adsorbent and ion-exchange materials in Resion are "totally insoluble and non-toxic."[3] No cases of constipation reported in three clinical series of more than 250 patients.[1-3]

*Available* **IN 2 PLEASANT-TASTING DOSAGE FORMS**

*Resion—for simple diarrhea.* Polyamine methylene resin 10%; Sodium aluminum silicate (synthetic) 10%; Magnesium aluminum silicate (synthetic) 1.25%.

*Resion P-M-S—for infectious diarrhea.* Resion; plus Polymyxin-B 125,000 units; Phthalylsulfacetamide 1.0 Gm.; Methyl Paraben 1.33%; Propyl Paraben 0.33%; Butyl Paraben 0.1%; in each tablespoonful (15 ml.)

*Dosage: RESION*.....1 tablespoonful hourly for 4 doses; then every three hours while awake.

*RESION P-M-S*....1 tablespoonful hourly for 3 doses; then 3 times daily. Infants—the same schedule as above, but in teaspoonful doses.

*Supplied:* Resion is supplied in bottles of 4 and 12 fluid ounces;
Resion P-M-S in bottles of 4 fluid ounces.

REFERENCES: 1. Weiss, J.: K.A.G.P. Journal 2:33, 1956. 2. Gabroy, H. K., and Selsman, G. J. V.: Amer. J. Dig. Dis. 20:395, 1953. 3. Lichtman, A. L.: Exper. Med. & Surg. 9:90, 1951.

*Products of Original Research*

NATIONAL

THE NATIONAL DRUG CO.
Philadelphia 44, Pa.

R-2700/57

# relaxes both mind and muscle

***for anxiety and tension in everyday practice***

- well suited for prolonged therapy
- well tolerated, relatively nontoxic
- no blood dyscrasias, liver toxicity, Parkinson-like syndrome or nasal stuffiness
- chemically unrelated to phenothiazine compounds and rauwolfia derivatives
- orally effective within 30 minutes for a period of 6 hours

*For treatment of* **anxiety and tension states and muscle spasm**

# Miltown®

*2-methyl-2-n-propyl-1,3-propanediol dicarbamate—U.S. Patent 2,724,720*

*Tranquilizer with muscle-relaxant action*

WALLACE LABORATORIES

SUPPLIED: *400 mg. scored tablets—200 mg. sugar-coated tablets*
USUAL DOSAGE: *One or two 400 mg. tablets t.i.d.*

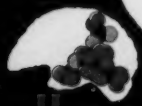
*Literature and samples available on request*

CM-3707-R4

*WOLF & WOLFF*

*HUMAN GASTRIC FUNCTION*

**now** "... *care of the man rather than merely his stomach.*"



# "Milpath"

Miltown® + *anticholinergic*

## controls gastrointestinal dysfunction

## because it cares for the man

### At the cerebral level

the tranquilizer *Miltown* in "Mil*path*" controls the psychogenic element in G. I. disturbances. (Miltown does not produce barbiturate loginess or hangover.)

## as well as his 'stomach'

### At the peripheral level

the anticholinergic, *tridihexethyl iodide*, in "Mil*path*" blocks vagal impulses to prevent hypermotility and hypersecretion.

**for** duodenal ulcer • gastric ulcer • intestinal colic spastic and irritable colon • ileitis • esophageal spasm G.I. symptoms of anxiety states

*prescribe:*
1 tablet t.i.d. at mealtime and 2 at bedtime.

"Milpath"

Miltown® + *anticholinergic*

Formula:

Miltown® (meprobamate) 400 mg. (2-methyl-2-*n*-propyl-1,3-propanediol dicarbamate) U. S. Patent 2,724,720

tridihexethyl iodide 25 mg. (3-diethylamino-1-cyclohexyl-1-phenyl-1-propanol-ethiodide) U. S. Patent 2,698,325.

**WALLACE LABORATORIES** New Brunswick, N. J. *Literature and samples on request*

# *On the Utilization*

# of Oatmeal Carbohydrate

One of the distinguishing characteristics of oats is its relatively lower content of carbohydrate and its higher content of protein, when compared with other whole-grain cereals.[1] An important feature of the carbohydrate of oatmeal is its mode of utilization by the body.

Because it consists mainly of slowly available polysaccharides with relatively minor amounts of dextrins and simple sugars, the carbohydrate of oatmeal does not tend to cause a sudden, sharp increase in blood sugar. Instead, the complex carbohydrate molecules gradually release glucose into the blood stream and thus provide energy on a more sustained basis. Oatmeal is therefore logically recommended as an excellent breakfast dish for diabetics.[2]

The carbohydrate content of oats is almost completely digested and utilized. Cellulose, or cellulose-like material, representing only about 2 per cent of rolled oats in the dry form, provides a gelatinous residue which serves advantageously as nonirritant bulk in the lower intestine.

Whether eaten for therapeutic reasons during illness or for a good sturdy breakfast every day, oatmeal and milk (skim or whole) assures high biologic quality protein, vitamins, and essential minerals, in addition to ease of nutritional utilization.

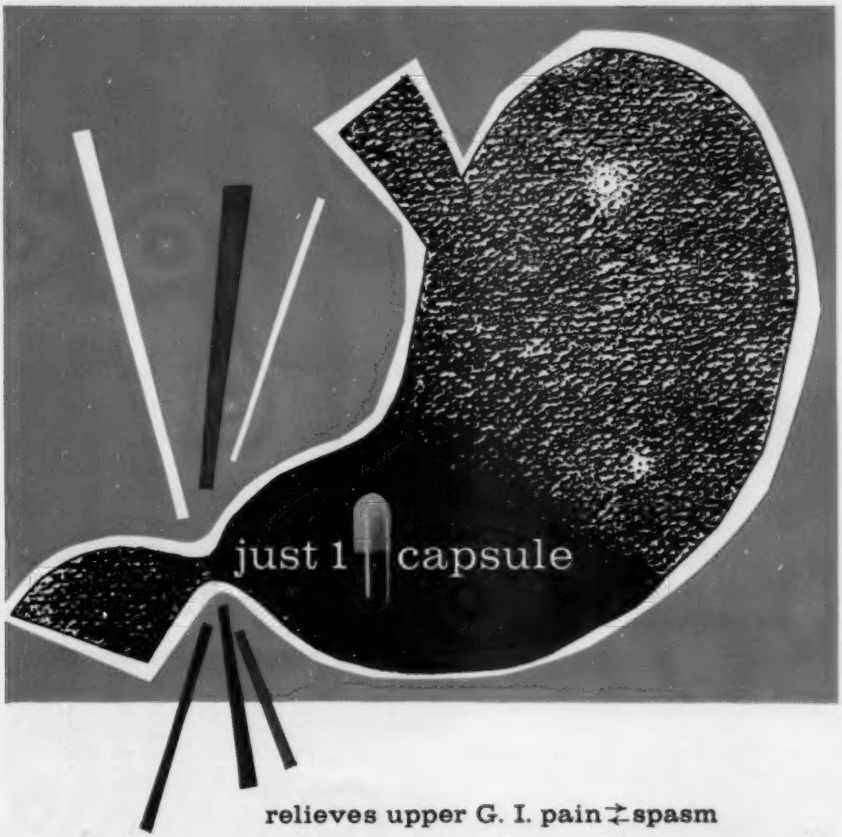
Quaker Oats and Mother's Oats, the two brands of oatmeal offered by The Quaker Oats Company, are identical. Both brands are available in the Quick (cooks in one minute) and the Old-Fashioned varieties which are of equal nutrient value.

**IN GASTROINTESTINAL AFFECTIONS.** Oatmeal fits well into the dietary management of gastrointestinal disease. It is bland, mechanically nonirritating, low in residue, yet high in its contribution of available protein, vitamins and minerals.


1. Kent-Jones, D.W.: Modern Cereal Chemistry, ed. 3, The Northern Publishing Co., Liverpool (1939).

2. Heupke, W., and Kolb, A.: Mechanism of the Beneficial Action of Alimentation of Oats in Diabetes, Deutsche Ztschr. Verdauungeskr. *13*:111 (1953).

The Quaker Oats Company
CHICAGO




**relieves upper G. I. pain ⇄ spasm**

**usually in 10 minutes**

**visceral eutonic**

# DACTIL®

**PLAIN AND WITH PHENOBARBITAL**

- normalizes visceral tone and motility
- does not interfere with digestive secretions
- avoids "antispasmodic" side effects
- prescribed q.i.d. for gastroduodenal and biliary spasm, cardiospasm, pylorospasm, biliary dyskinesia, gastric neurosis and irritability

**DACTIL is the *only* brand of N-ethyl-3-piperidyl diphenylacetate hydrochloride.**

LAKESIDE

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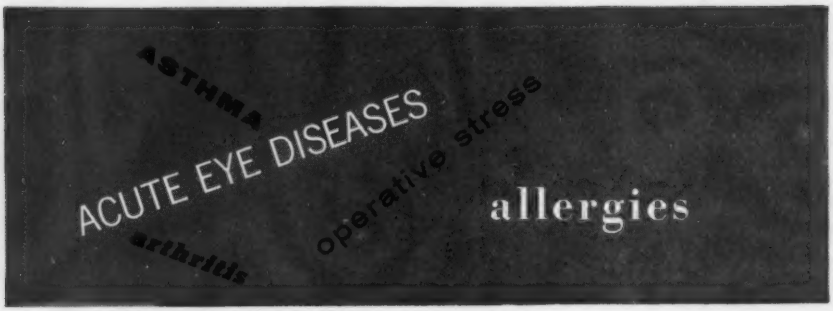
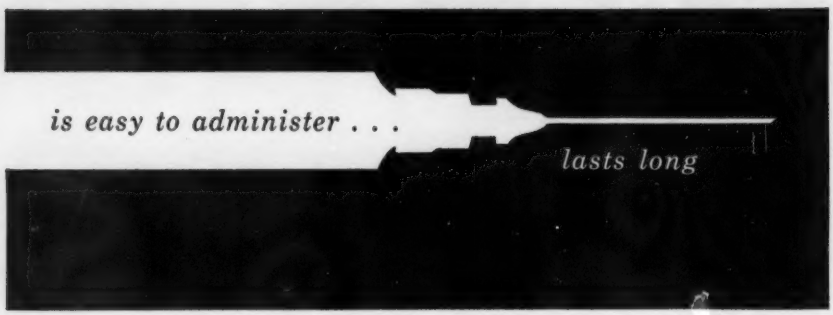



*Economical treatment of the many conditions amenable to ACTH therapy*

# CORTROPHIN*-ZINC†




Use Cortrophin-Zinc to provide relief in the 100 or more conditions amenable to ACTH therapy. It's economical because each injection lasts at least 24 hours in the most acute cases, to 48 or even 72 hours in milder cases. It's easy to handle, too, because it is an aqueous suspension which flows freely through a 26-gauge needle with no preheating. Clinically proven in thousands of patients, an advance in ACTH therapy.

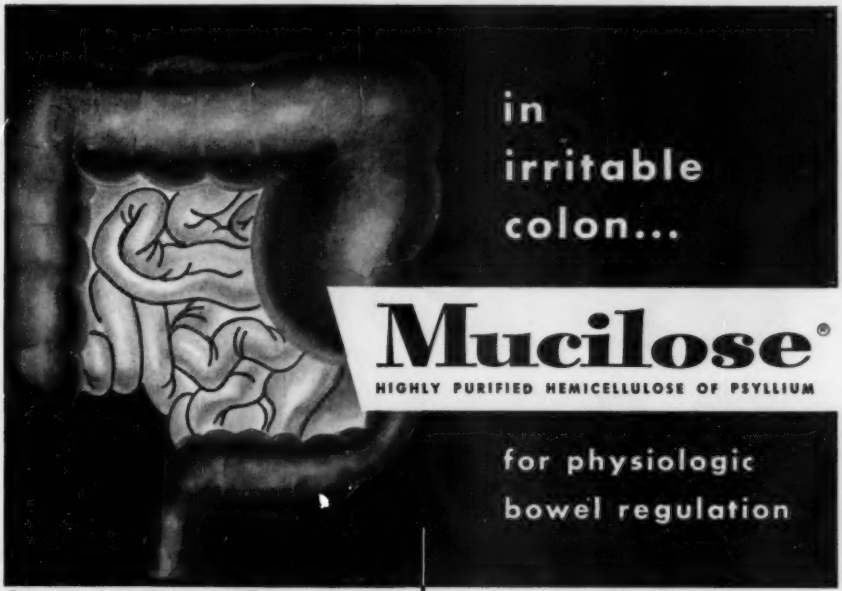
Available in 5-cc vials, each cc providing 40 U.S.P. units of corticotropin with 2.0 mg zinc.

*T.M.—Cortrophin

†Patent Pending. Available in other countries as Cortrophine-Z.

**CORTROPHIN-ZINC**
*an Organon development*

Organon INC.
ORANGE, N. J.



in irritable colon...

# Mucilose®

HIGHLY PURIFIED HEMICELLULOSE OF PSYLLIUM

for physiologic bowel regulation

## Variety of Dosage Forms for Flexibility in Prescribing

- **MUCILOSE COMPOUND TABLETS,** Mucilose with methylcellulose; *bottles of 100 and 1000*

  ***Greater Bulk • Smaller Dosage Convenient • Easy to Swallow***

  Also
- **MUCILOSE SPECIAL FORMULA FLAKES or GRANULES**

  and
- **MUCILOSE CONCENTRATED FLAKES** *in tins of 4 oz. and 1 lb.*

**In constipation**

Mucilose should be taken with 1 or 2 glasses of water.

Winthrop LABORATORIES
NEW YORK 18, N.Y. • WINDSOR, ONT.

Mucilose, trademark reg. U. S. Pat. Off.

**"...the important problem of constipation" in irritable colon should not be overlooked.**

**"It is imperative that the patient stop taking laxatives."**

**"The routine use of a hydrophilic colloid such as...Mucilose...is often gratifying, and never contraindicated in irritable colon syndrome even with diarrhea."[1]**

**MUCILOSE** produces bland peristalsis-stimulating bulk resulting in soft, demulcent stools. This is due to its tremendous water-binding capacity (50 times its own weight[2]). Conversely, when taken with small amounts of water, Mucilose restores the normal water balance in the diarrheal phase of colitis by binding loose stool and reestablishing normal fecal colloid.

1. Straughn, R. A.: *Jackson Clin. Bull.*, 13:83, Aug., 1931.
2. Gray, Horace, and Tainter, M. L.: *Am. Jour. Digest. Dis.*, 8:130, Apr., 1941.

**MUCILOSE** *For Physiologic Bowel Regulation*

# *not as simple as* "APC"...

The search for an analgesic preparation superior to APC has culminated in the development of FALGOS TABLETS. After extensive pharmacological research, alkaline substances – which speed absorption[1] and minimize the side effects of acetylsalicylic acid[2] – have at last been successfully combined with APC in a stable, pharmaceutically elegant tablet.

**Faster pain relief . . . better gastric tolerance**

The distinct advantages of Falgos were recently demonstrated in a controlled study[3] of hospitalized patients. In 1528 of 1694 administrations (90.2%), complete relief was provided within 10 to 30 minutes without side effects. Some degree of relief for aches and pains in a variety of mild to severe somatic conditions was experienced by 98.9% of all patients studied.

In another series of studies in which buffered and unbuffered APC tablets were compared, the buffered material was shown to be definitely superior to the unbuffered tablets from the standpoint of faster, more effective pain relief and better gastric tolerance.

**Each Falgos Tablet contains:** aspirin 3 gr; acetophenetidin 2 gr; caffeine ⅓ gr; and a balanced proportion of aluminum and magnesium hydroxide.

**Indications:** Pain of headaches, neuralgias, neuritis, muscular aches and pains, discomfort of the common cold; minor aches and pains of rheumatism and arthritis; before and after dental procedures and tooth extractions.

**Dosage:** 1 or 2 tablets with water, repeated every three hours if necessary.

**Supplied:** Bottles of 15 and 40 tablets.

# FALGOS®

***ANALGESIC-ANTACID TABLETS***

1. Lolli, G., and Smith, R.: New England J. Med. 235:80 (July 18) 1946.
2. Schnedorf, J. G.; Bradley, W. B., and Ivy, A. C.: Am. J. Digest. Dis. 3:239 (June) 1936.
3. In press.

**For samples, write: AMERICAN FERMENT CO., INC., New York 18, N. Y.**

# Add to the team for safer bowel surgery

**SULFATHALIDINE**

PHTHALYLSULFATHIAZOLE

**SULFATHALIDINE** used preoperatively gives you the comforting assurance that intestinal bacteria have been almost entirely suppressed, that danger from fecal contamination is greatly lessened. Maximum local effect is insured because of low systemic absorption. Given postoperatively, **SULFATHALIDINE** promotes early healing, speeds convalescence. **SULFATHALIDINE** is also useful in treating ulcerative colitis.

**CREMOTHALIDINE®** is a palatable suspension of **SULFATHALIDINE.**

MERCK SHARP & DOHME

DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

## when he smokes too much . . .

Whenever indigestion results from habitual overdoing in any form (excess of cigarettes or alcohol, dietary indiscretion, nervous tension), symptoms can be quickly alleviated night and day with Gelusil and the new formulation Gelusil-Lac.

**To avoid daytime distress:** Gelusil provides the sustained action of magnesium trisilicate and specially prepared aluminum hydroxide gel to restore and maintain gastric pH within normal range. Because it does not overneutralize or alkalize, Gelusil avoids the twin dangers of acid rebound and systemic alkalosis.

To prevent middle-of-the-night attacks: In Gelusil-Lac, the proven antacid action of Gelusil is combined with sustained buffering effect of specially prepared high protein (low fat) milk solids to prevent gastric pain at night.

**Nonconstipating:** The specially prepared aluminum hydroxide component in Gelusil has a low order of chemical activity. Thus formation of astringent – and constipating – aluminum chloride is minimal.

**Dosage:** 2 Gelusil tablets or 2 teaspoonfuls Gelusil liquid two hours after eating or when symptoms are pronounced. Each tablet or teaspoonful provides: 7½ gr. magnesium trisilicate and 4 gr. aluminum hydroxide gel. Gelusil-Lac: At bedtime, one heaping tablespoonful stirred rapidly into one-half glass (4 fl. oz.) of cool water. (Provides equivalent of 4 Gelusil tablets.)

# Gelusil®/Gelusil-Lac

**WARNER-CHILCOTT**
100 YEARS OF SERVICE TO THE MEDICAL PROFESSION